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VOL. I.—42ND YEAR SYDNEY, SATURDAY, FEBRUARY 26, 1955 No. 9

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THE TEETH AND FOOD.[1]

By F. W. CLEMENTS, M.D.,
Institute of Child Health, Sydney.

THE Annie Bromhall Cunning Lectures were endowed in 1942 by Mrs. Cunning "for the promotion and encouragement of medical education and the dissemination of knowledge" concerning nutrition. In expressing my deep appreciation of the honour of delivering the eighth lecture of the series, I am also conscious of the responsibilities this involves. My predecessors have set a high standard which will be difficult to emulate. The subject is an important one and no apology is needed for its inclusion. Even if the founder of these lectures had not expressed the hope that it would be included in the series, recent interest justifies its inclusion. Posterity may decide that the current decade was a critical time in the conquest of dental caries; my concern is to do justice to its importance.

Caries in History.

It would seem that man has suffered the scourge of dental decay from the earliest times. Some skulls of Neanderthal man, who roamed this earth some fifty thousand years ago, have frankly carious teeth. Most textbooks on dentistry draw attention to the fact that the Egyptians from about 3000 B.C. onward suffered from caries; of particular interest to us is the fact that the prevalence was much greater in the wealthy upper classes, who appparently enjoyed a much more luxurious diet than their slaves. Through the history of western Europe we find repeated references to toothache arising from caries. The high level of development of Etruscan dental workmanship, which produced all manner of gold bands and other fitments for artificial teeth, is clear indication of a need arising out of teeth lost through disease.

Kriskos, who has examined teeth from different periods of Greek history, provides some interesting facts. Skulls from Greece of about 2300 B.C. were caries-free; by 1700 B.C. 8% of skulls contained carious teeth; this figure had risen to 10% 1000 years later, and another 1% to 2% had been added 1000 years later. By 1300 A.D., another 1000 years later, the figure was 20%; by 1900 the incidence had risen to more than 50%, and in the opinion of Dr. Kriskos, the caries rate was still rising. It is significant that in this Grecian series little difference was found in the structure of the teeth originating from prehistoric to mediæval times. However, teeth from recent periods and the present times show a significant increase in developmental defects, as revealed by deficient histological structure of the enamel and dentine.

[1] The eighth Annie B. Cunning Lecture on Nutrition, The Royal Australasian College of Physicians, delivered on November 24, 1954, at Sydney.

The numerous studies of recent and contemporary primitive people living in their traditional manner and eating their traditional food, and of people of the same ethnic groups who have gravitated to the outposts of civilization, have provided interesting results. Undoubtedly the most energetic worker in the field was the late Weston Price, who paid a flying visit to this country in the 1930's to study selected groups of aborigines. His investigations extended from Switzerland to the Gaels in the Outer and Inner Hebrides, to the Eskimos of Alaska, to the ancient civilizations and their descendants in Peru and in the Amazon Basin, and to many groups in the Pacific. Although not all dental scientists agree with Price's conclusions, they cannot dispute his findings in respect of the condition of the teeth of these groups. While caries was relatively rare in those people living their traditional life, it was frequently rampant in those who had substituted the trade-store foodstuffs for their traditional diet. Price also reported changes in the shape of the dental arches in the second and subsequent generations of these people.

The island of Lewis in the outer Hebrides provided further evidence of contrasting conditions. In 1937, King found that 28% of primary school children and 24% of secondary school children, born and bred in the country districts of the island, were free from caries, compared with 2% and 7% of those who had lived all their life in the town of Stornoway.

Although we have come to accept dental decay as a concomitant of civilization, recognizing that it has been aggravated in every society where it has occurred when the cultural pattern has become more sophisticated, there is ample evidence that minor degrees of caries occurred long before our modern food habits were evolved. A limited number of studies suggest that even in contemporary times (that is, the last twenty-five to thirty years) the incidence of caries is rising; this suggests that, in these localities, the ætiological forces are increasing in intensity. In many other places the incidence in children has fallen. I shall refer to this again.

Theories of the Cause of Caries.

Ideas about the causes of dental decay based on scientific observation and supported by clear logical thinking are of comparatively recent origin. For at least 3000 years man believed that caries was due to the gnawing of worms. This idea, in addition to being very old, was apparently almost universal. It was accorded credence in ancient Egypt and Mesopotamia, right through Roman times and the Middle Ages, and existed in India, China, pre-Colombian America and Oceania. Townend reports the recent personal experience of a woman in an isolated district of England, who declined to have her child's carious tooth extracted, on the grounds that she could obtain from the local chemist something to destroy the worms that were the cause. Because of its antiquity, it is highly unlikely that there was a rational basis for the origin of this idea; however, once extractions became common practice, from Roman times onward, the occasional protruding pulpal tissue at the apex of a tooth, which appeared to wriggle on exposure to the air, and which disappeared into the tooth owing to contraction, must have served to perpetuate this ancient belief.

Pierre Fauchard, who practised his calling in Paris in the first half of the eighteenth century and who has been called the founder of modern dentistry, was the first to cast serious doubts on this theory. However, he had no sensible single suggestion to offer as an alternative, nor does it seem that he was very interested in this theoretical consideration. His interests lay almost entirely, as did those of the French school of that period, in improving dental techniques. References to the worm theory continued to appear in text-books for the next fifty years.

To John Hunter must go the honour of laying the foundations of the modern scientific approach to dental caries, through his careful study of tooth structure. His conclusions were based on two components: first, that teeth were different from any other tissue, including bone, and consequently their diseases were different; and second, that they had a "living principle", through which the tooth secured nourishment while sound, but which failed to operate when it was deceased. His "living principle" stood in the way of a sound correct conclusion as to the cause of caries. In spite of the fact that he observed that the surfaces of the teeth which became carious were those where food and tartar collected and "the juices are pressed and there stagnate", he finally concluded that "owing to a deficiency of nourishment from some fault in the vascular system" caries "is a disease arising originally in the tooth itself".

Hunter's mistake influenced the thinking in England about dental caries for some considerable time, and because of the apparent hopelessness of the situation no attempts at prevention were made. Joseph Fox, who lectured at Guy's Hospital in the beginning of the nineteenth century, expressed the rather hopeless conclusion that "This delightful secret [the discovery of some mode of prevention of caries], although it is pretended to in the advertisements of every quack, we can only expect to acquire when the Philosopher's Stone and the grand panacea have been obtained".

The nineteenth century saw America seize from France leadership in dental technique and from England leadership in the conceptual developments in dentistry.

Parmly, an American trained dentist who travelled extensively in England and Europe, and who was fully acquainted with the works of Hunter, Fox and others, rightly concluded that caries started externally, and that the mistaken ideas of Hunter and Fox could be explained by the fact that "the aperture in the enamel through which this disease finds its admission, is in many cases so exceedingly minute as to escape detection of all but those who are aware of its true cause". In his pamphlet, published in London in 1818, he postulated that a "chymical agency" arises in the relics of food which collect around the teeth, and that this substance corrodes their structure. It is interesting to note the similarity between Parmly's writings and those of Henry Jeanneret, the author of the first Australian book of dentistry, which appeared in 1830. The similarity is too close to allow us to ignore the possibility that Jeanneret had come under the influence of Parmly.

Not only did Parmly give the "man in the street" some hope that if he preserved a high degre of oral cleanliness caries would be reduced, he also provided the dental profession in America with its own dynamic theory of dental caries which culminated in the work of W. D. Miller some sixty years later. Miller was an American dentist practising in Berlin, who worked in the laboratory of Koch in his spare time. In 1890 he published his book, "The Micro-organisms of the Mouth", in which he postulated the "chemico-parasitical process", stating that caries consisted of "two distinctly marked stages; decalcification, or softening of the tissues, and dissolution of the softened residue". He affirmed that "decalcification was due to acids which are derived from the particles of amylaceous and saccharine substances which lodge in the retaining centres and there undergo fermentation".

Modern Developments in Support of the Chemico-Parasitic Theory.

Since Miller's time many workers have added their contributions to expand and develop his theory. The foodstuffs which have been singled out for the most extensive studies are refined white flour and sugar, more particularly the latter. The justification for intensifying investigations along these lines has come from the following four sources: the results of numerous field studies, which have pointed strongly to the importance of sugar consumption; the effects of diets low in carbohydrate on the caries rate in diabetic and other children under strict dietary control; studies of the structure, pathology and biochemistry of the plaque which forms on teeth; and advances in enzymology, which have shown how acid can be produced by bacterial action from carbohydrates.

Field Studies.

Through the late 1920's and 1930's Weston Price was reporting his observations, to which I have already referred, and from which he had drawn the conclusion that the high consumption of refined foods, especially white flour and sugar, as substitutes for the rougher, often raw, traditional foods was the cause of the dental decay and deformities.

In 1932 His Majesty's Ship *Carlisle* had returned from its visit to Tristran da Cunha, and Sampson had reported that the people living in this remote island in the South Atlantic had excellent teeth, with scarcely any caries. Because of their isolation, with an almost complete absence of outside trade, the diet of these people was determined by what they could grow or could gather from the sea. The diet consisted of fish, potatoes, milk and vegetables; since no wheat was grown on the island they did not have bread or flour unless it was obtained from a passing ship; likewise what little sugar they used came from the same source.

Effects of War-time Diets.—The next important stimulus came from the observations made in eleven European countries that the teeth of the children during World War II had significantly less caries than in the pre-war and post-war periods. Although the first reports came from England, the most intensive and most informative observations were made in Norway. With the exception of an influential group of British workers, to whom I shall refer later, most dental and nutritional scientists believe that the reduction in the consumption of refined white flour and sugar during the war was the cause of the fall in the incidence of caries.

The Local Study.—A field study which has created considerable interest in this country and abroad is the one which has been continuing at the homes for children controlled by the Youth Welfare Association of Australia. Mr. L. O. Bailey, a Sydney business man, who was the founder of the association, devised a diet based on his reading and personal experience, on which some 80 odd children have lived from early infancy to their present ages, which range from five to twelve years, the majority being between nine and twelve years of age. This is the group which Dr. N. E. Goldsworthy and his co-workers at the Institute of Dental Research have studied so painstakingly over the past seven years. The diet of these children consists of wholemeal bread, wholemeal biscuit, wholemeal porridge, wheat germ, fruits (fresh and dried), vegetables (cooked and raw), cheese, eggs, milk, fruit juices, nuts, nutspreads, vitamin concentrates, and for sweetening, on occasions, honey or treacle. This diet is adequate in all the known nutrients; but the most important fact is that the children have not had access to refined white flour or sugar from birth onward. Dr. Goldsworthy's group reported in 1952 that 63 out of 81 children, whose ages ranged from four to nine years, were free from caries. The average number of decayed or filled teeth per child was 0·58, which was lower than that for the children of approximately the same age group in three New Guinea villages investigated by the same institute in 1947.

Diabetic Diets.—In 1939 Boyd reported a much lower incidence of caries in a group of children with controlled diabetes, compared with the same age groups in the general population. This observation has been repeated by other workers, and the regimen is now used, apparently with success, by many dentists to control rampant caries in children and adolescents.

Modern Developments in Bacteriology and Biochemistry.

Numerous workers have repeated Miller's original experiments, in which he showed that, *in vitro*, the organisms found in the mouth could ferment starch and cane sugar with the production of acid. However, workers soon discovered that the buffering and diluting action of the saliva kept the pH of the resulting solution in the cavity of the mouth above the critical pH to produce caries. Further, if teeth were placed in an acid solution *in vitro*, general erosion of the tooth resulted, and not a typical carious lesion.

The part played by dental plaques on the surface of the teeth in concentrating acids has received considerable attention. Wild, who studied the plaque under a stereoscopic microscope, reported that it grows on the surface of artificial and natural teeth within three days of cleaning. At first is it a thin, glistening membrane, but soon builds up into a rough, fur-like mat which changes from day to day. It is composed of proteins, apparently those found in the saliva, and a mass of interwoven organisms. The method of attachment of this plaque to the tooth is not clearly understood.

The effectiveness of the plaque as a barrier between solutions in the oral cavity and the surface of the tooth depends on the concentration of oral solutions. In the resting phase the saliva is free of sugar, but after a meal rich in sugar the concentration of sugar may rise to 2000 milligrammes per 100 millilitres and half an hour may be required for its clearance. Stephan has shown that dilute solutions of sugar are accompanied by the production of acid reactions on the surface of plaques, with little penetration of the plaque by the sugar. During the sucking of confectionery, a concentrated solution of sugar develops around the teeth; some of this sugar apparently penetrates through the plaque to the surfaces of the teeth. The pH of the plaque or of a carious lesion can drop from 7·0 to below 5·0 in a few minutes and remain there from fifteen minutes to two hours, depending on the concentration of the sugar and the individual characteristics of the mouth. When the concentration of the acid reaches a pH of 5·0 or lower the enamel is decalcified. These minute attacks are repeated every time concentrated carbohydrates gain access to the interior of the plaques.

Fundamental researches in biochemistry which have revealed the anaerobic phase of carbohydrate degradation by enzymatic action, provide an explanation of how acid is produced in the mouth. Some of these enzymes are released by the microorganisms, others appear to come from the saliva. Tom Spies and his group in Alabama have made an important contribution in this field as a by-product of their studies of pellagrins. Having confirmed that their subjects were practically free of caries, as are most pellagrins, they went on to show that there was a close positive correlation between the nicotinic acid content of the saliva and the extent of dental caries. Deficiency of nicotinic acid in the body is the cause of pellagra; it is a component of co-enzyme I, which operates in an intermediate stage of carbohydrate metabolism, being essential for the production of lactic and pyruvic acids. There seems to be much evidence to suggest that lactic acid plays some role in the production of caries.

The Proteolytic Theory.

This brief review of contemporary developments would be incomplete without reference to the proteolytic theory of caries production. It is obvious that the protein matrix of the tooth as well as the inorganic material, must be destroyed in caries. The advocates of this theory suggest that caries is primarily the result of invasion of the sheaths of the enamel prisms by proteolytic bacteria. Although originally developed by Preiswerk in 1907, this theory has been revived in the last decade by Gottlieb in America and by Pincus in England. The latter has suggested that sulphatase enzymes, which can be produced by Gram-negative bacilli present in carious material, release the SO_4 ions and so lead to the formation of sulphuric acid from the polysaccharide present in the organic material. The dissociated hydrogen ions thereupon link with the PO_4 ions of the enamel substance, which leads to its destruction. Pincus's work has been largely on dentine. He makes no attempt to link up clinical observations with his theory —for example, the pronounced fall in caries in Europe in World War II.

The Effects of Systemic Nutrition on Tooth Structure.

Criss-crossing the orderly development of the chemico-parasitic theory during the last thirty-odd years has been

the counter theory that the widespread development of caries in children's teeth is primarily a problem of defective nutrition, which results in imperfect formation of their teeth. The more the hypoplasia, the more, in general, the caries.

Lady May Mellanby developed this hypothesis about 1918, as a result of her examination of the teeth from puppies which her husband had used for rhachitic studies. They found that if bitches were given, during pregnancy, diets which led to the development of rickets in the puppies, the same puppies had a greater tendency to develop carious teeth.

Early in the 1920's Lady Mellanby commenced a series of surveys of the teeth of selected groups of children in England. The first studies were made of children aged from five to fourteen years who were living in institutions in Birmingham. In addition to their normal institutional diet, they were given special dietary supplements of vitamins *A* and *D*. It was found that the progress of dental caries in the permanent teeth had been significantly retarded in the experimental group, compared with the controls.

In 1928, in association with Pattison, a dentist, Lady Mellanby found that the addition of vitamin *D*, in the form of irradiated ergosterol, "checked the initiation of new carious points, diminished the spread of old carious points and arrested the infective process in many carious teeth".

The most spectacular observations were made in the post-war period, when a number of teams, all of which have included Lady Mellanby, found great reductions in the caries rates in groups of children. They found that "in general, dental health of children examined in 1943 was much better than in 1929 and in 1945 there was further improvement". In 1949 they reported that the progressive improvement found in two previous surveys had also been observed in 1947. The rate of improvement between 1945 and 1947 had been more rapid than between 1943 and 1945.

When seeking an explanation for these improvements, the authors restricted their comments almost exclusively to the changes in the composition of the diet in England under war-time rationing, which had ensured a much larger consumption of foodstuffs rich in calcifying items —namely, calcium and vitamin *D*—by both pregnant women and children. In view of the great support being accorded the "sugar theory" by many groups of workers, especially in America, as early as 1940, it is to be regretted that Lady Mellanby made no reference in any of her papers to sugar consumption in England during the war, and no attempt to relate the "sugar theory" to her own findings. Her work has been severely criticized by both dentists and nutritionists; the former quarrel with her techniques for detecting hypoplasia and dental caries, and the nutritionists hold that she did not take all dietary facts into consideration.

Support for the concept that the level of total systemic nutrition, as opposed to one process (as advanced by Lady Mellanby), may have an effect on dental caries has been offered by Sognnaes, of the Harvard School of Dental Medicine. His views are based on two sets of data. He has made a detailed analysis of the results of 27 war-time surveys which had been made in European countries, covering about 750,000 children. He found that a reduction in dental caries either did not occur or was inconsistent during the first years of the two World Wars, compared with the occurrence of a pronounced and uniform reduction during the last years of the wars. Secondly, the major reduction in caries occurred, not during the wars, but at varying periods after, the time interval depending on the age of the children studied. As Sognnaes points out, sugar rationing was introduced soon after the wars started, and terminated in some countries soon after they finished. This was particularly so in Norway, where the caries rate in young children in the recent post-war period has remained low.

Sognnaes draws special attention to the differentiation reduction in caries rate between seven-year-old and three-year-old children in Norway in 1946. In the former the reduction in caries in the permanent teeth, compared with pre-war figures, was 8%, whilst for three-year-olds there was an 80% reduction in caries in the deciduous teeth; yet as Sognnaes points out, during the year 1945 both sets of teeth were subjected to the same environmental factors in the mouth. These figures indicate a greater reduction in caries in those children who were born towards the end of the war and whose teeth were being formed *in utero* during a period of severe dietary restrictions. The constant time lag suggests that, in the formation of caries, systemic factors which operate during the formation of the teeth may be as important as the environmental conditions in the mouth in the post-eruption stage.

The other contribution made by Sognnaes is from his animal experiments. He found that caries-susceptibility of rodents and sub-human primates is greatly influenced by nutritional changes instituted during the period of tooth development. He fed rats and monkeys on diets containing a large amount of sugar, and failed to find a relationship between the incidence of caries and the duration of exposure of the teeth to the diet after tooth eruption. A significant increase in susceptibility to caries was noted in those animals whose teeth were developing while the parent animals were on the diet. The diet seemed to affect the structure of the teeth, making them more susceptible to caries.

Dr. Goldsworthy recently reproduced a graph supplied by Mr. Grandison, of Cambridge, which shows the percentage of decay present in the temporary teeth of five-year-old children in Cambridge, England. The basic data were collected by this one dentist from his public health work, from 1911 to 1952. The incidence of caries before World War I was 35%; this dropped sharply towards the end of the war to about 12%, rose again in the 1920's, but only to 20%, dropped in the middle of World War II to 10%, and in recent years has risen to 15%. Thus there has been a progressive fall from 1911 onward, some, but not all, of which has occurred during the two wars; the interesting fact is that the post-war rise on each occasion was to a level far below the pre-war figures. Throughout all this period there has been a steady but pronounced improvement in the over-all nutrition in England, and especially in the nutrition of pregnant women.

Sugar and Caries.

The belief that sugar or sweets may cause dental caries has long lingered in the minds of men. Aristotle in about 330 B.C. asked: "Why do figs when they are soft and sweet produce damage to the teeth? Perhaps because their vicious softness causes small particles to adhere to the gums and insinuate themselves into the dental interstices where they easily become the cause of putrefactive processes." In 1695 John Locke, in his famous "Treatise on Education", advised "no sweets or titbits ever to be allowed at odd times". From time to time during the nineteenth century and the early parts of this century, physicians and specially those interested in the welfare of children, have given warning that too many sweets lead to caries. However, it has taken the results of the unplanned war-time mass experiments and the studies on small groups of children to bring the importance of sugar into the spotlight.

I should now like to spend a few minutes reviewing what we know about the forms in which sugar is consumed and the order of importance of each of these in the production of caries. The present consumption of sugar in Australia is about 130 pounds per head per year, having risen from about 117 pounds per year in the 1920's and around 110 pounds early in this century. The ways in which the sugar is distributed through foodstuffs has also changed. In 1932 householders purchased directly about 74% of the sugar; the balance (26%) went to manufacturers of processed foods and to the breweries. In the last four years manufacturers have used about 47% of the sugar consumed in Australia—proof of the extent to which factory-processed foods have replaced domestic cooking and food preparation. The domestic consumption of sugar has remained fairly constant over the last thirty years at about 70 pounds per head per year. Most of the actual increased

total consumption has occurred in the sugar used for manufactured foods. In the last twenty years, the sugar used in jams has increased by 30%, that in confectionery by 50%, and that in cordials and jelly crystals by more than 300% (a large part of the last-mentioned is used in the manufacture of heavily sugared cordials). The amount of confectionery produced in Australia, at present, would provide approximately three ounces a week for every man, woman and child.

There are no figures in Australia for the consumption of confectionery by children. The only data are those produced by Widdowson from her pre-war survey of the food consumption of some 900 children in England. She found that from 12% to 18% of the Calorie intake of boys and 12% to 16% of that of girls were provided by sugar. About one-third of this was as sugar, and the remainder was fairly evenly distributed between confectionery, jam, biscuits and puddings. If sweets and chocolates are taken together, children aged two years ate three ounces per week and those aged seven years five ounces.

A very significant fact in Widdowson's survey is that children aged one year obtained 13% of their Calories as sugar. Dietary surveys in England and Australia have revealed that about 13% of the Calories consumed by households were supplied by sugar; thus it would seem that infants have the same level of sugar intake as the rest of the household, except for some pre-adolescent children and adolescents who obtained up to 18% of the Calories as sugar. In other words, from infancy, children are taught the taste of sugar and are apparently quickly brought up to the same level of sugar consumption as the rest of the family. Mothers preparing dishes for infants and young children make a habit of tasting the food for sweetness and general palatability. Their own level of appreciation of sweetness soon becomes the level of the infants' taste. In this way a constant high level of sugar consumption is perpetuated in the community.

In the production of caries, the total amount of sugar eaten is apparently not as important as the time and method of consumption, if we accept the results of the study made at a mental institution at Vipeholm, Sweden, from 1946 to the present time. The experiment cannot be reported here in detail, but briefly seven groups of men and women were given sugar-rich diets over four years. The sugar was incorporated in food, or drunk in a solution, or eaten as chocolate or toffee. The groups of subjects who consumed the toffees between meals unquestionably had the greatest increase in new caries. Sugar in the form of caramels and chocolates also eaten between meals appeared less cariogenic than the toffees, but was still well above the bread with even considerable amounts of sugar added. The latter method of giving added sugar (even in amounts equivalent to that in the confectionery) seemed to have caused little or no increase in tooth decay beyond the control levels.

This study would suggest that if children stopped eating sugar between meals, in the form of confectionery, chocolates and perhaps biscuits, there would be a significant reduction in the incidence of caries.

All investigations to date suggest that caries does exist in the presence of what is otherwise an excellent state of nutrition; some children consume enough sugar at wrong times to produce caries, without producing changes in their general nutrition which can be measured by any existing techniques. However, when 18% of the Calories are obtained as sugar, other nutrients must be replaced by the sugar, or else the children would show excessive weight gains. Although sugar has the same calorific value as other carbohydrates, it lacks the vitamins which are found in most other carbohydrate-rich foods—for example, white flour.

Carbohydrates require certain vitamins of the *B* group for their complete combustion; Australian white flour contains sufficient vitamin B_1 to ensure the satisfactory metabolism of the carbohydrate in the flour. Sugar differs from flour, in that it must obtain the necessary vitamins for its metabolism from other foods. If a high percentage of the Calorie needs is regularly obtained from sugar and there is not a deliberate attempt to increase the vitamin B_1 intake, a state of chronic vitamin B_1 deficiency can develop.

It is well known that children who regularly eat relatively large amounts of sweets, particularly between meals, have poor appetites and disturbed digestions. In other situations these symptoms have been attributed by some workers to vitamin B_1 deficiency. Numerous pædiatricians over the last hundred years have recognized various disturbances which arise as the result of excessive intake of carbohydrates. I have only to instance Robert Hutchinson's "dyspepsia of the second dentition", or "habitual indigestion" found in text-books of the last century.

At what level of sugar intake normal nutrition gives way to these disturbances is unknown. It would seem to be when the Calories derived from sugar are between 12% and 18% of the total Calorie intake. However, I want to stress again that rampant caries can exist in children whose nutrition is satisfactory by all clinical standards.

Recapitulation.

Even were I capable, I do not consider it appropriate tonight to argue the merits of the various contributions that have been made to the current theories of dental decay. At this stage I propose to review briefly the various ideas about the cause of caries against the scientific and social environments current at the time when each was advanced. Before the advent of the scientific method, man took an analogy from every-day events around him; a worm apparently caused decay in an apple, in timber left on the ground—why should a worm not cause decay in his teeth? This view persisted until the rise of modern anatomy, with its emphasis on both macroscopic and microscopic examination of structure as the basis of theoretical discussion. It is true that John Hunter misinterpreted his observations, possibly as John Greenwood suggested, because of his lack of experience with a wide range of dental conditions. However, Hunter, the anatomist, did lay the foundations for the advances of the nineteenth century, which culminated in Miller's chemico-parasitic theory at the end of that century. Although the ideas incorporated in this theory had been gathering during the nineteenth century, their consolidation into a theory, the general features of which could be proven, had to wait until the great advances in the techniques of bacteriology, initiated by Pasteur, had been applied in the laboratory by Koch as a routine procedure. It is surely significant that Miller worked in Koch's laboratory. The elaboration of the ways in which the acids, recognized by Miller, are produced by microorganisms from the carbohydrates remaining in the mouth, had to wait until the appropriate fundamental discoveries had been made in the fields of biochemistry.

It was inevitable that a nutritional theory of the cause of caries should have arisen after the epoch-making discoveries of Eijkman and Funk. The initial proposition was made by Mrs. (now Lady) May Mellanby only five years after McCollum and Davis had established the existence of a fat-soluble vitamin, which at that time was a mixture of vitamins *A* and *D* (the separation was not effected until some four or five years later). Reviewed at this distance, it seems to have been inevitable that the suggestion that the level of calcifying factors in the diet (namely, calcium and vitamin *D*) should determine susceptibility to caries, should have come from workers in England at the time when it did.

Then, finally, as I have already said, it took a war with its food restrictions to focus attention on the significance of sugar in the ætiology of caries.

I am fully aware that this review errs in the direction of over-simplification with all the defects of such procedures. However, I do find support and comfort in the recent memorandum on dental health of children issued by the Child Health Committee of the British Dental Association. The Association has recognized two prime factors in the ætiology of caries in children: (1) the diet-

of the mother during pregnancy and especially the intake of fermentable carbohydrates, which can influence the structure of teeth, and (ii) the sugar consumption of the child in the post-eruptive phase, which provides the substratum for the bacterial production of acid.

All these theories of the ætiology of caries have omitted another factor, which in terms of prevention is probably the most important. I refer to the social *milieu*. It is about this that I now want to speak.

The Social Milieu.

The facts I have been discussing up to this point are now called by some writers the "internal logic" of science —in this connexion dental science and some allied sciences. There are many examples from the history of science to show that the mere accretion of scientific facts (including medical facts) does not ensure their application. The social and intellectual environment of the period must provide the conditions favourable to their application. This influence has been given a variety of names by writers, including "external forces" and the "social *milieu*". So constant is the association between the "internal logic" of science and the social *milieu*, that the opinion expressed last year by Lilley is axiomatic of all developments and is confirmed both within scientific development and in the history of man's conquest of his environment. He wrote that "any scientific development becomes possible only when both the internal and external conditions are ripe". For at least a decade the "internal conditions" have clearly indicated ways to prevent the development of dental caries in children. Nevertheless, except for isolated experiments like those of the Youth Welfare Association of Australia, a few similar institutions abroad and the efforts of individual families, the achievements in the community to date in the prevention of caries are no better than might be expected if the scientific advances of the last thirty years had not occurred. I suggest that the social *milieu* is at present unfavourable, even hostile, to the application of the scientific facts presented tonight. The justification for this conclusion lies in the community's attitude towards caries.

Although no surveys have been made in this country to determine the community's attitude to caries, we have the experience of the School Medical Branch of the New South Wales Department of Public Health. So poor was the response of parents to notifications of uncomplicated caries in their children that the Branch has discontinued sending notifications. The personal experience of a large number of dentists is that many adults fail to seek treatment for carious teeth until overtaken by toothache. These people maintain the same attitude about their children's teeth. Too many parents think the primary dentition unimportant and later carry this thinking through to the second dentition.

This general lack of interest could arise from the community's feelings about caries as a disease. Ackerknecht, the medical historian, is satisfied that decisions of society rather than objective facts determine what shall be considered diseases. We, the professional people, may call caries a disease, but this is apparently not enough. Ackerknecht gives some interesting examples of communities' attitude to a disease. Pinta, one of the treponematoses akin to syphilis, is so common among certain tribes in Mexico and Colombia that the few healthy men not manifesting the disease are considered abnormal. In other words, the communities do not accord pinta the status of a disease.

Nearer at hand we have the attitude of the people in New Guinea to what is apparently chronic malaria. Victims of this condition are expected to continue working with some self-medication and perhaps a few hours' sick leave. There chronic malaria is not accorded the status of an illness.

In recent times medical science has extended the definition of disease to include many departures from normal structure and function which do not endanger life; but society's definition seems to be still limited to those conditions which, untreated, threaten its continued existence. It is this fear which spurs to action the great majority of people who seek our advice. Caries, particularly uncomplicated caries, is not in this category; it evokes no fears to motivate action.

The people who seek regular care for their teeth and those of their children are motivated by an entirely different set of forces; a sound set of teeth has for them a high appeal. Although these people are the exception today, they could be the forerunners of a decisive movement, and the forces which have motivated them could provide the motive for the mass of the people. Much depends upon whether society will feel strongly enough about the æsthetic qualities of a sound set of teeth to change its attitudes to the offending food stuffs.

The Control of Caries—Dentistry or Sociology.

To ask children to forgo confectionery and similar cariogenic foodstuffs, which have a high prestige value, which are frequently used as rewards, which have a pleasing taste, and which provide gratification of the oral phase of development, is unrealistic so long as parents continue to eat sweets in an unrestricted manner, and so long as society refuses to provide adequate motivation —high esteem and social acclaim of a set of sound teeth. Although a handful of children have been conditioned to a diet almost completely lacking in sweetness, in the absence of motivation it is virtually impossible to teach the mass of children a new concept or to persuade them to change their food habits.

Campaigns for the control of dental caries cannot use the psychological appeal which has formed the basis of practically all nutrition work. Most of the discoveries in the science of nutrition have led to drives for better human diets, with campaigns to "eat more" of this or that foodstuff. Most of these foods are delectable and have a high prestige value in our society. To eat them and to make sacrifices to give them to our children for their better health have a strong appeal to the average man and woman, which has been used directly and indirectly by health educators.

The control of dental caries, through restricted and controlled sugar intake, in the absence of community pressures, has proved more than the majority of the population can accept. There is some evidence that it is too threatening to the personal liberty and the emotional security of the community at large. Already efforts are being made to develop more acceptable alternatives for the control of caries. The demands for fluoridation of water supplies are increasing, and scientists are searching for an anti-enzyme which can be incorporated into confectionery, tooth-paste and what have you, on the hypothesis that the anti-enzyme would prevent the formation of acid from sugar. These are legitimate scientific projects; but as alternatives to controlled sugar intake they represent the capitulation of the scientist and the public health administrator to community attitudes.

Earlier I pointed out that each advance in dental science towards the solution of the problem of caries had to wait for the solution of a fundamental problem in an allied science. It would seem that advance beyond this point in the mass control of caries by dietary means will depend upon a significant increase in our knowledge of community attitudes—a study which lies in that borderland where dentistry impinges on sociology, called by some social medicine (or social dentistry). The deciding forces will not be scientific facts, of which we have ample, but human reactions to these facts, of which we know so little. I want to conclude with the words of Clyde Kluckhorn, who said that the study of human reactions to situations and problems constitutes "the great intellectual and emotional challenge of the second half of the twentieth century".

Acknowledgements.

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HAY FEVER AND THE GENERAL PRACTITIONER.[1]

By C. T. PIPER,
Adelaide.

LET us begin this account with some case reports.

Towards the end of October, 1953, a man (Mr. H.) suffering from hay fever consulted me. He was an old patient of our clinic; had had successful pre-seasonal desensitization in 1951 and 1952. He came to us in mid-August, 1953, with recurrence, and was given peptone intravenously, which brought on asthma; he then went off and drove in the Redex Trial, and remained free of his hay fever amid the bull dust of that trip. A few days after his return it started again, and by October 22 had reached disabling proportions; on that day he was given an intradermal injection of a new extract containing the product from one milligramme of grass pollen; this raised a small histamine weal on his arm, which went away in one hour. Four days later he was given a similar extract from 10 milligrammes; this raised a weal five-eighths of an inch in diameter with two inches of erythema about it. He reported on that day that his hay fever was much better. He was given further injections of 10 milligrammes on October 28 and on November 2, 4 and 6, by which time he was free of hay fever and we were quite sure that the injections had produced the result. The showery weather had gone, we were having typical dry November hay fever weather, and there was no other change of circumstance. The patient reported at the end of the season that he had had no hay fever at all since his first injection of the new substance.

On November 2 a middle-aged man (Mr. E.) came to the out-patient department with severe hay fever. He was given a one-milligramme dose of the grass pollen extract, with a moderate reaction. He reported on November 5 that he was still having hay fever, and I gave him 10 milligrammes; he developed a large weal about two centimetres in diameter and an acute flare about six centimetres in diameter and within a quarter of an hour had generalized acute histaminosis with some laryngeal obstruction. He was given adrenaline, also "Benadryl" intravenously, which controlled this condition within a few minutes, although he remained flushed and uncomfortable for an hour. Three weeks later he came in to me and said: "If I get my hay fever again can I have another of those treatments?" I told him that he could, but said that I felt that I would not like to have it. He said, "I would sooner have that than my hay fever", and went on to say that he had had no hay fever since the injection, though he was still taking "Phenergan" at night. He had no more hay fever last season.

On November 19 a young woman (Mrs. S.) came to the out-patient department in considerable distress with hay fever. She was given an injection of one milligramme intradermally, which was repeated on November 23 and 30; she reported on November 23 that her hay fever had disappeared before she got home on November 19, and she remained well until the end of the season.

On November 9, 1953, a middle-aged woman (Mrs. S.) was sent from the country, seeking relief from spring asthma. She had then had it for a month, and she had had it every spring for the last three years. On that day she was given an injection of one milligramme. On November 12 she seemed somewhat better, and was given five milligrammes; she then had to return home, and I gave her some of the substance, with instructions for use, to take to her own doctor. She reported on January 26 that she had not had any need to have further treatment after she went home, and she had had no more hay fever and no more asthma.

These patients are typical of some 50 whom we treated with this new product in the spring of 1953; in all we treated 67 patients; some of the results are unknown.

It is this treatment which is now under experiment and trial in certain factories by the Department of Medicine of the University of Adelaide, a matter which found publicity in the *Sunday Advertiser* in mid-August. That newspaper announcement is the genesis of this paper. It was thought at the time that, this publicity having been given to a new treatment, it was a proper courtesy to the medical profession that it should be informed of what is going on, so I approached the South Australian Branch of the British Medical Association for an opportunity to make an interim report. This is necessarily an interim report and may be a report of a squib; but it is still, I think, a matter of considerable interest that we have been able to get these results at all.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on September 30, 1954.

HOW COMMON IS HAY FEVER?

Hay fever has news value in this community. It has news value because a very large number of people suffer from the disorder, and a very large number of people think they suffer from it. Just how common it is one does not know; there is an impression abroad that hay fever is on the increase, and we certainly seem to see many more patients in practice now than we used to do. This may be because people and doctors are more aware of the condition; it may be because we now have antihistamines and people must have a prescription to get them; it may be that there is a true increase. I have asked some doctors who have been in family practice in the same area for a long time whether they think there has been a true increase, and most of them say that they think the increase is in the awareness rather than in the incidence; but it is the opinion of oto-rhino-laryngologists that many more people come to them seeking relief from hay fever now than twenty years ago. My own impression before coming into the practice of allergy in 1951 was that there was much more of it then than there had been twenty years before, and I am fairly sure that that increase has been associated with the spread of rye grass over the plains and of fog and phalaris in the hills.

It is not easy to make a true assessment of how common it is; but at the Long Range Weapons Establishment, where pollen exposure is maximal and where anybody who is going to get hay fever will get it, one in 13 of the people working has volunteered for treatment under this experiment. Quite a number of these will probably not have true hay fever; but even if we write off some 10% and bring the number down to one in 15, that will mean that there are approximately 30,000 potential hay fever sufferers in the metropolitan area alone. Now 30,000 people is a lot of people, as one can see at the Adelaide Oval on any Saturday this month. How many of them suffer from crippling hay fever I cannot say, except that approximately 150 new patients seek relief from grass pollinosis in our various clinics every year; these are but a fraction of the whole.

HOW IMPORTANT IS HAY FEVER?

Sufferers from severe hay fever will tell you that hay fever impairs their efficiency by anything up to 25% during the season, and has a serious effect on their personal relationships. They will cordially agree with the statement that "I have hay fever and my wife suffers from it". A percentage of these people suffer from disabling asthma, and when people start to get asthma they are starting to get a serious disorder. I think it is probable that about 20% of the people who come to me for relief from hay fever suffer also from grass asthma at some time in the season, usually at the height of it.

Of course the majority of victims suffer from hay fever as a nuisance only; but a disease which is so common is a matter which must be of interest to all of us, and to the family practitioner in particular. To the allergist it is important in three ways: in the first place, of course, because it gives him a considerable part of his livelihood; in the second place, because it is pure allergy, and the study of it should give him some indication of what the processes behind allergy are; and in the third place, because a great deal of his year is so extremely occupied with pre-seasonal and seasonal treatment of the disease that he is not able to find time to think on those aspects of his specialty. Therefore it is, I think, important that a simple treatment for hay fever should be found which will pass it back to the general practitioner in all its stages, and that is one of the objects of the experiments which have led to this paper. Hay fever is a major disease in some ways, but mostly it is a minor disease and should not require specialist treatment.

What is Hay Fever?

Properly defined, hay fever is seasonal allergic rhinitis due to grass pollen, with which may be associated allergic bronchitis, allergic dermatitis and urticaria. The characteristic of it is œdema of the mucosa of the nose with serous exudate containing eosinophile cells; the symptoms are obstruction of the nose, sneezing, watery discharge, lachrymation and itching of the palate and ears. Between attacks the mucosa may be normal, it may be pale, glazed and slightly swollen, or it may have the wash-leather appearance characteristic of the chronic allergic state. Allergic rhinitis may be due to any dust containing protein. However, the term hay fever is applied to allergic rhinitis of other origin, so we define the following three principal types of the disease: the sporadic type, usually due to animal emanations or to insect-borne pollens; the perennial type, usually due to environmental, domestic or occupational dusts, or to the products of saprophytic bacteria; and the seasonal type, due to wind-borne pollens. It is this seasonal type with which we are concerned tonight.

Allergy and Allergic Reactivity.

Hay fever, then, is an expression of allergy. I think I should define the term allergy for you. To be allergic literally means to work oppositely. The study of allergy is the study of disturbances of physiological mechanism due to abnormal reaction to foreign substances, especially to normally non-inimical foreign substances, or to non-inimical doses of harmful substances. These non-inimical substances are usually common to the environment of man and have been so since time immemorial. The phenomenon of abnormal reaction without clinical manifestation is known as sensitivity, while abnormal reaction with symptoms is known as hypersensitivity; and although hypersensitivity may be removed by various means, sensitivity remains and may develop into hypersensitivity at any time. This last tendency means that no improvement gained by a desensitization method is permanent, and that there is no such thing as cure.

From the viewpoint of the allergist there are two kinds of people, the allergic and the non-allergic. Skin tests show that at least 20% of people have sensitivity to normal environmental non-inimical substances; probably only 20% of these have hypersensitivity and have clinical reaction at consulting room degree at some time in their lives. So the allergist divides his subjects into two kinds, the latent and the reacting allergics. These again may be divided into three classes—the pollen allergics, the domestic allergics and the animal allergics; the division is not very distinct, and people may be allergic to all three types of allergen. Tonight we are principally concerned with the reacting pollen allergics.

Allergic Reactivity.

Before passing on to discuss pollen allergy or pollinosis, as it is properly called, I think a word on allergic reactivity is in season. It is one of the "not understood" things about allergy that equal exposures to allergen do not necessarily produce symptoms in the same person. There is some other factor operating to determine clinical reaction, some factor which raises or lowers the threshold of allergen concentration necessary to produce disease. It is probably that factor which holds the key to the problem of asthma. One part of that factor, the part played by emotion, is well known to all of you—it is a fascinating and mysterious facet of the disease. It does not play a large part in pollinosis, although many people are still willing to reiterate Beard's mistake of 1876, when he estimated that 100,000 people of the United States had hay fever, and labelled it "essentially a neurosis". I think it is quite established that the threshold of reaction to air pollen content varies little in each individual; and anyone who has had it will, I think, readily agree that the hay fever produces the mood, not the mood the hay fever.

Natural History of Hay Fever.

I think here a word about the natural history of the disease is opportune. Going about his work, every allergist finds many people who show high degrees of sensitivity to grass pollens, but have no symptoms from it, and many who once had symptoms for many years, but now no longer suffer from them, although they still give a strongly positive reaction to skin tests. Study of case cards kept for many years gives a sort of composite card which suggests that pollinosis is a self-limited disease of very long duration, much as follows. There is a stage of preclinical sensitivity, a period of increasing hypersensitivity, a plateau of maximum reaction, and then a *diminuendo* fading to a stage at which sensitivity remains but clinical reaction is gone. Each phase seems to occupy about five years; the process may apparently start at any time of life and the onset of hypersensitivity and clinical reaction may seem to be quite abrupt; but such onsets are usually due to environmental changes which by virtue of heavier exposure reveal a hitherto unrevealed hypersensitivity.

A history of fifteen years is common, and a history of twenty-five years is rare. This is only an average statement, but it means that you can cheerfully tell your patients that they will eventually grow out of the disease, a thing which you cannot justly tell to the parents of an asthmatic child.

Pollinosis in South Australia.

The hay fever pollens in South Australia are as follows: (i) September to November 31: grass pollens: early barley grass, wild oats, *Poa annua*, middle-phalaris, fog grass, late rye grass (from mid-October). (ii) November 31 to Christmas: grass pollen as house dust. (iii) November to early March: plantain, couch grass, yellow broom. (iv) March and April: autumn flowers—dahlias, daisies, chrysanthemums *et cetera*. (v) May and June: American ash. (vi) August: almond blossom time; unknown pollen, possibly false caper (most of these people are not reactive to almond blossom); Iceland poppy in homes. The season is a month earlier in the far north, a month later in the south-east and southern hills. Saltbush and castor-oil plant cause a few cases. Grass sensitivity is a group sensitivity, flower sensitivity is somewhat more specific.

We are fortunate here in South Australia that we really have only one season when hay fever is epidemic, and that is the grass pollen season. In Europe and North America they have an epidemic pollen season due to the deciduous trees in the spring, and a grass pollen season in summer; and they also have in America an autumn season due to the pollens of the rag weeds, which are very commonly spread throughout the continent. This combination makes it possible to have pollen hay fever all the year round in some of the southern States. We have abundant flower pollen here, but fortunately practically all our native and garden flowers and garden escapes are pollinated by bees or other insects and the pollen is heavy and sticky and is not easily carried in the air. Only one is important, and that is the plantain.

The pollens of grasses and the insignificant plantains and some less common weeds are not carried by insects, but rely on random scattering by the winds. This means a great wastage of pollen, and therefore a great production and a very light pollen which is designed to float in air for long distances. It is wind-borne pollen that causes epidemic hay fever, and that is why we have more hay fever on dry days and north-windy days than we do in damp weather, when the rain carries the pollen down to the ground or makes it heavier by dampening it so that it sinks of its own accord.

The grass pollen season here usually starts about mid-August, with pollens that are blown down from the north, and with some other pollen which comes about almond blossom time; I do not know what this pollen is, but it may be the grass called *Poa annua*, or it may come from the weeds of the false caper series. That is an unfinished business that I must work out next year. The season starts locally about a month earlier in the far north, and ends about a month later in the hills and in the south-east; it usually goes on in Adelaide until just before Christmas. It persists after the grasses have gone

thoroughly to seed, because the pollen gets into houses and remains there for a few weeks until it has rotted; so that hay fever, which in November is a diurnal disease, in December mostly comes on at night after the patient has gone to bed, and has the characteristics of a house dust allergy.

The flower pollens usually cause hay fever only when people walk amongst them or pass by in very close proximity, or stand under the ash trees in May or June. When flowers are used to decorate houses and places of entertainment, the pollen drops, dries and becomes part of the normal house dust, and so gives rise to hay fever as a domestic allergy.

What Can Be Done about Spring Hay Fever?

There are three basic principles in the treatment of allergy, and one bad treatment: (i) avoidance of the allergen; (ii) drug therapy; (iii) the bad treatment, nasal surgery; (iv) specific hypo-sensitization.

Avoidance of Allergen.

Avoidance of the allergen is practically impossible. The north winds of November will see to that; any patient who is grass-hypersensitive in this community will be attacked on such days. However, it is possible to reduce the incidence by elimination of grass from the footpaths, the gutters and the paddocks next door. It is this local pollen which is principally responsible for severe hay fever and for the inconvenience from which most people suffer. One of the reasons why so many more people seem to suffer from hay fever may be that there has been such a sudden spread out into new housing areas, where unmade footpaths and roads and vacant blocks are filled with grasses; it is also pertinent to this increase that the rye grass, originally planted in the 1920's, has spread throughout the agricultural areas and has replaced the barley grass, which is not so potently allergenic nor so profuse in its pollen, in many of these vacant blocks about the metropolitan area. Anybody with hay fever should, of course, keep the house shut and remain inside on north-windy days; if it is necessary to drive through grass areas, the motor-car windows should be kept closed. However, I am afraid that for people in the new areas there is little escape, and I would strongly advise any man who is contemplating moving to these parts or to the hills to have a skin test before he does so, and also to see that his wife is tested, for women suffer much greater indignities from these things than men. It is said that the best treatment for hay fever is to have a good sense of humour; but it is very difficult for a woman to be amused when she has a perpetually red and weeping nose.

A large number of our patients come from these new areas, where they are attacked for the first time in the first spring after they go there to live; they have not been conscious of their pollen hypersensitivity while living in heavily built up areas with fully paved roads and footpaths.

Medication.

You are all familiar with antihistamines, and their administration is the treatment of choice for mild and occasional hay fever; but the perfect antihistamine remains to be made. There is considerable individual idiosyncrasy, and most of these drugs have some depressing side effects; to be really effective in severe hay fever they have to be taken in doses which are as disabling and unpleasant as the disease itself. My own idea of their proper use involves the use of a long-acting sedative antihistamine at night, such as "Phenergan", and of a mild one by day, repeated as necessary; for this I use "Allecur" or "Diatrin"; unpleasant side effects from these can usually be relieved by the use of amphetamine or ephedrine. "Benadryl" is, I think, the most potent of the antihistamines, but not many people willingly take it. I have sufficient belief in it to use it as a therapeutic test when I am in doubt as to whether I am dealing with an allergic state or not.

Local medication helps; there are many drops, all of them rapidly losing their virtue. I favour "Fenox" and "Antistin Privine", probably more from habit than anything else. It is a good tip that they are sometimes more effective introduced into the conjunctiva than *per nares*; antihistamine cream in the nostrils helps many people.

Nasal Surgery, Ionization, Cauterization.

Nasal surgery during hay fever is absolutely contra-indicated and is of value only to improve the airway between the seasons. Ionization and cauterization certainly give relief for a time, but I think most rhinologists agree that these treatments are to be avoided; both depend on mucosal destruction, both result in scarring and produce an irreversible lesion. If a patient comes to me who has had more than four treatments by either or both, I take a very dim view of his prospects of relief from my bag of tricks.

Treatment by Methods which Reduce Hypersensitivity.

Treatment by desensitization, or more properly hyposensitization, is the basis of the allergist's therapeutic armament. It is really a long-term symptomatic treatment; it does not cure the allergy and does not diminish reactivity, but it simply raises the threshold over which allergens must climb to promote a reaction. It does this by giving "one hair of the biting dog", then two hairs, then four hairs, and so by gradual progression to 10,000 hairs, experience having shown that to be a protective level for most people. It means a long series of injections, and most of you know the story. It is a difficult business, and more of an art than a science, and only experience can get good results from it; there are many nuances in it, and I think it is essentially a job for the specialist who sees enough of it to understand them. Against pollens it is satisfactory only in its temporary results. In the hands of those who understand it, I think 85% of patients can expect to get satisfactory relief for that season; but in the hands of the inexpert who do not know the details, relief for 60% is probably all one can expect. Such relief as is gained is temporary and treatments must be repeated annually or continued on a perennial basis. It is unsatisfactory, in that it must be begun when the patient is well and most remote from his disease, and he must have anything from 30 to 40 injections over a period of five months, each of which involves an attendance of at least half an hour at the doctor's rooms. Any of these injections may give rise to uncomfortable, distressing and even dangerous reactions. The treatment is wasteful of money, and very wasteful of what is more valuable, time. The usual starting dose is one microgramme, and this must rise by slow degrees to 10 milligrammes; even at one microgramme many patients get an unpleasant reaction.

There are intraseasonal treatments. The accepted one, which seems to work in about one case in five, is to give minimal doses over three weeks, starting at one microgramme and increasing by one microgramme to 10 microgrammes, repeating these as symptoms recur; even these doses can cause reactions in the season.

In 1926 Phillips in America described an intradermal treatment with pollen protein solution, which gave him in 29 patients "monotonously successful results"; his objective was to raise a weal the size of the patient's hand which endured for twenty-four hours, and he claimed to get his results from three or four treatments; he doubled the dose at each treatment and gave about 10 doses through the season. This treatment has never been accepted by allergists, and I should think that non-acceptance is due to the reactions which it must have provoked; in the days before antihistamines for intravenous use, these must have been rather fearsome things. I found this reference early in July, and the results bear such striking similarity to our own that I must report it here and give it credit.

Peptone Given Intravenously.

There is, however, another older intraseasonal treatment, which seems to give good results in about one case in 15; it is mentioned in all text-books, only to be condemned as useless. That treatment is the intravenous administration of peptone. There is no doubt that when it works, it works well; gradually increasing doses will give relief at

TABLE I.

Patient.	My Estimate of Relief.	Patient's Estimate of Relief.	From *Questionnaire*: Relief Obtained.	Dates of Injections.	Number of Injections.	Comment.
M.	50%	75%	Three days after.	November 24, 26, 27.	3	Was very crippled with hay fever. Minimal dosage. Lives at Stirling—the mid-season.
E.	80%	75%	Within 24 hours.	December 4, 7.	2	Late in season, but result very rapid and very effective.
G. ..	Nil	Nil	——	November 6, 9, 11.	3	Could be inadequate treatment.
D.	100%	75%	After first injection.	October 29; November 3, 6.	3	Very satisfactory.
P.	60%	66%	After first injection.	November 4, 11, 20.	3	Had considerable relief from each injection.
A.	Nil	Nil	——	——	—	Failure—our worst and most difficult hay fever.
I.	100%	75%	Day after.	December 2, 4.	2	A dubious result, as this was a sudden attack that could have passed off as quickly.
K.	80%	75%	30 hours after.	November 3, 17; December 3.	3	Each dose gave him 12 days' relief.
MacN. ..	80%	88%	One week later.	November 9, 12.	2	Takes his hay fevef very seriously—reliable witness.

the sixth or seventh dose, and relief can then be regained by about two doses when symptoms recur. My impression is that the relief lasts for a much longer time than that gained by formal hyposensitization, which must be repeated every season to be certain of results. It actually worked for me the first time of using, and that was the beginning of this paper.

I have always been puzzled as to how non-specific peptone could work, and it was this puzzling that gave me the idea that specific peptone should work better. Last year I asked Mr. F. Draper if he would peptonize some pollen for me, and he fortunately made a 10% solution. I rather expected to receive a substance of low allergenic value which I would use intravenously.

Development of New Technique.

It was while we were testing this quality by intradermal injections of decimal dilutions that it was observed that some patients were getting better of their hay fever, and the first patient whose story I related is the first one in whose case we really confirmed this impression. We went on treating people, and I made in December, 1953, a review of 52 cases of which the results were known; this indicated to me that we had attained a satisfactory result in 41, and that the average improvement gained by those 41 patients was in the nature of 80%; over the whole group we had had an average improvement of over 60%. These results were so impressive that I decided that I would send out some *questionnaires*; this I did in January, 1954, and the analysis of the replies gave the following confirmation.

Results with New Technique.

We sent out something over 50 *questionnaires*; at the end of three weeks we had had 48 returned, three of which were discarded as not understood by either the patients or us, so I finally analysed 45 *questionnaires*. Table I is taken from a typical sheet made in the compilation of that analysis. Thirty-seven of these 45 patients acknowledged a relief of 50% or more. The average improvement of the 45 patients was 64% by their own estimate, and the average improvement in the 37 patients with more than 49% relief, by their own estimate, was 77·5%; the average number of injections given to all patients was 3·8. This confirmed my December estimate. It was a matter of interest that grass pollen dermatitis disappeared under treatment with this method. We had previously had nothing that had been of any value against it.

Twenty-eight of these people had their last relieving injection before November 21, which is when the rye grass pollen starts to wane. Fourteen state that they had relief immediately or within twenty-four hours. There were 15 failures to my knowledge among 67 patients, and in all but three of these I could find a reasonable explanation of the failure. It is important that most of the patients who had any subcutaneous injection of this substance had no relief.

General Reactions.

The bogy of all these hyposensitization treatments is that of reaction. We have had such reactions; they take the form of general histaminosis with flushing, heat, tachycardia and mild laryngeal œdema; they respond quickly and satisfactorily to adrenaline and the intravenous injection of an antihistamine, and we have not a great deal to fear of them. The patients who have such a reaction usually get a good result from the treatment. We have had only one reaction of the shock type, and that was with flower pollen in a person of very low skin sensitivity. The elimination of these reactions is necessary before the treatment can be handed out to those who are unfamiliar with it or not prepared to use it with great care; we are at present working on this matter. One thing apparent to us is that all doses must be given with good tuberculin syringes; these are very hard to come by.

It is to be noted that Phillips used 0·02 milligramme of pollen to produce his big reaction; we use one to 10 milligrammes to produce our small one. Our idea at present is to raise a weal about 1·5 centimetres in diameter with an erythematous halo about five centimetres in diameter; 10 milligrammes of pollen protein is the final dose given in preseasonal desensitization. Our ideal adequate reaction is about that which one would expect in the preseasonal period from an intradermal injection of 0·1 of "A" strength extract—that is, 0·001 milligramme injected into a strongly sensitive person. It is our impression that four such reactions are adequate. If five do not give relief we believe that the treatment is likely to be a failure.

"Toxoid" Nature of Peptonized Pollen.

It seems, therefore, that we have succeeded in producing a substance similar to the toxoids of diphtheria and tetanus, a substance which has lost its toxicity but retained its immunizing capacity. Therefore, for convenience we call our product "toxoid", although I think that name is probably wrong and that it will have to be changed if the substance ever comes into general use.

It would be more exact to say that we have removed allergenicity but retained antigenicity.

Experiment with Other Peptonized Allergens.

These results with the grass pollens were so encouraging that we decided to apply the same process, treatment with hydrochloric acid and pepsin, to other allergens, so we peptonized some plantain pollen and some flower pollen and for good measure some house dust; we hoped thereby to gain some verification of results and further experience against the 1954 pollen season. We had similar results, and we found that we were able to abolish flower pollen asthma with a great degree of certainty, to relieve plantain

hay fever and dermatitis as with the grass pollens, and to get quick control of house dust rhinitis and asthma in some cases.

Animal Emanations.

In April a case occurred which gave us an opportunity to try the material with animal emanation.

G., a technician at the Institute of Medical and Veterinary Science, had recently come back to working with guinea-pigs. He had worked with them before and had suffered from rhinitis and itchy hands when he handled them; but for the two months before he came to us he began to have asthma every night after he handled them by day. He gave a positive reaction to guinea-pig hair; when he wore a respirator at work his asthma disappeared, when he left his respirator off his asthma came back again. We considered this a proven case. We peptonized some guinea-pig hair of an unknown percentage, and gave him a testing dose on a Tuesday; we gave him adequate reaction doses on Wednesday, Thursday, Friday and Monday, and on Tuesday he went back to work with his guinea-pigs without the respirator. He had no asthma that night and has remained free of it until recently. As he is grass-pollen sensitive, we suspect a compound asthma.

We have had other encouraging results with asthmas due to animal hair, but have had no such pure cases as this. I have a great hope that this treatment will enable every asthmatic boy to keep a dog. If that is so, then I have not lived in vain.

At the end of April we reviewed the situation, and found that with these other extracts we had a 60% adequate relief rate. This was fairly satisfactory, and we declared an interim while we prepared a complete range of peptonized extracts from all the usual allergens. These have now been in use for about two months, and we are still getting a 50% adequate relief rate or better, although we are doubtful about permanence, and there is a possibility that we are making some people worse. We have excluded grass pollens from this count; they will be on probation for the next few months in a separate schedule.

Effect in Atopic Dermatitis.

We have had particularly impressive results in dermatitis. We have previously had no specific treatment for pollen dermatitis, and we have not had a failure here in the few cases in which we have been able to be certain that pollen is the causative factor. The following fantastic case is illustrative of what can happen.

Mrs. L. was first examined on May 10, 1954; she was referred on account of dermatitis of the face, forearm and hands of thirty months' duration. The condition had been continuously present and active during that time, but for the last six weeks had been acute, becoming worse on Saturdays. The patient had had much treatment from dermatologists, but had had no continued relief. The only protein sensitivity found was a mild reaction to flower pollens, and she had been picking chrysanthemums each Friday. The rash was worse on the day after the test. On May 11 she was given two injections, a total of 0·6 milligramme of pollen derivative. There was little local reaction; but for the next few days she had some generalized œdema with erythema of the hands and legs and some increase of the rash. The rash then began to recover, and by May 21 all activity had gone. By June 1 only a few scabs remained, and these were quite gone by June 10. She avoided flowers during that time, but as a general rule had not handled them more than once a fortnight during the preceding two years. As the patient lived on the seafront, she had no garden.

On September 20 the patient reported with an acute recurrence which had appeared on September 18; she was given 0·5 microgramme of the same substance with practically no reaction. All activity disappeared from the rash next day.

We have had two sufficiently encouraging results with egg, milk and gluten toxoids to think that we may have something that will help in infantile eczema. Some results in dermatitis have given us reason to doubt the need for specificity in this disease.

I think it should be noted here that we have obtained substances of similar allergenic potency by treating pollen with pancreatin and papain. We have not used them in treatment.

WHAT DO THESE RESULTS SIGNIFY?

I believe that there is no doubt that we have something in this treatment, but just what we have we do not know. There are certain things of which we are confident. We are actually confident that we have a quick answer to the intraseasonal relief of pollinosis in three out of five people at a late stage of the season. We feel that we have some answer to the general problem of desensitization; we think that apart from anything else, this treatment will be of use in indicating which of the substances causing positive skin reactions is actually the cause of the patient's asthma, and so be a valuable guide to formal hyposensitization. We also believe that there is a possibility that this treatment will so raise tolerance that the treatment can be started at 100 microgrammes instead of 1·0 microgramme. This would cut courses down by one-third.

We believe that it is probable that we have found a new tool for use in allergic research for those who know how to use it, for it is apparent that treatment which will change a man from a continuously and severely reacting allergic to a latent allergic in twenty-four hours by metabolic methods, as has happened many times, should facilitate investigation into the underlying metabolic changes which bring about the reactive state, the real basic problem of asthma.

Need for Research in Allergy.

In this respect I would point out that hay fever should be important to the pathologist. The pathogenesis of allergy is incompletely understood. The histopathology is clear, the chemico-physical pathology is partly so, but the pathogenesis is so obscure that there is still dispute as to whether it is a part of immunology or not. I confess that all these things are beyond my understanding; but I doubt that there is one theory of allergy which even conserves, let alone holds, water when critically examined in the light of clinical knowledge.

The study of allergy is made more difficult by the many facets in it. If those facets can be sorted out and appraised singly, then it seems reasonable to use this approach. For that purpose hay fever is practically pure hypersensitivity, while the young allergic child presents the other side of the picture in which hypersensitivity is minimal, but reactivity is at a maximum.

Sir Macfarlane Burnet has described poliomyelitis as the last frontier of infectious disease. Following his metaphor, I think it is fair enough to say that allergic disease is one of the least explored territories of pathology, and that our present investigations and knowledge have no more than reconnoitred its frontier. It seems to me that an understanding of its curiously ineffective and purposeless inflammatory processes and the way they are mediated as a psychosomatic process would make a notable contribution to the understanding of inflammatory disease in general. In this respect I would point out that many of the diseases against which man, by himself, has little effective defence are characterized by an allergic type of histopathology. Examples are acute rheumatism, arthritis, tuberculosis and poliomyelitis itself. I have a great respect for Selye's adaptation theory; but I cannot accept it as final on allergy any more than I can accept any of the other theories as final. That hypersensitivity to grass pollens is a failure of adaptation I admit; but so are lobar pneumonia and alcoholic gastritis, though in different degrees. The influence of cortisone on allergic inflammation is no more evidence of suprarenal exhaustion in allergy that it is of that condition in boils or pneumonia, in which states inflammation is equally suppressible by cortisone, provided the dose is adequate. I admit readily the psychosomatic element in allergic disease, particularly in asthma; but the *modus operandi* of that emotional nexus is quite beyond explanation unless one postulates an intermediate hormonal mechanism. Discovery of that mechanism may have an important bearing on all inflammations, a matter of obvious importance to the whole of pathology.

It seems to me that scattered through the world are a few people who are trying to sort out the basal facts of allergy. Amongst physicians there are two groups—the allergophiles, obsessed with the phenomenon of hypersensitivity, the outward evidence, and the allergophobes, to whom the whole business of allergy is anathema and the whole syndrome is just psychosomatic. This comforting theory of psychosomatism means that the physician has done his duty if he finds a frustration or conflict (common in everybody's life) and, having told the patient to "unfrustrate" or to "unconflict", dismisses him from his presence with pills and a bottle of medicine, and from his mind with the sense of duty well done. While these two groups guard the frontiers there is little hope that clinicians will promote expeditions into the hidden land; but if a treatment is found which will swiftly remove the allergic manifestation by obviously allergic methods, then perhaps the "phobes" will be shocked into belief and the "philes" jolted into looking beyond hypersensitivity for an explanation of the things with which they deal. Observation of allergists and their writing suggests that most of them would like to dive into the unexplored land, but they are so busy with their little needles they have no time to do it.

Conclusion.

All we claim at present is that we have had sufficient success to be certain that it is possible to detoxicate pollen protein and yet to retain its effectiveness as an antigen protective against pollinosis. Whether this can become the basis of a generally acceptable, simplified treatment remains to be proved in the field of large numbers. One hopes that it will be an acceptable treatment and usable by the family doctor. We have been lucky in many ways with these experiments, and it may be that we have just achieved a series of flukes; whether that is so or not will be proved by Professor H. N. Robson's department this spring in an experiment in three factories with a total of 10,000 employees.

Addendum.[1]

Since this paper was presented I have had Hansel's book, and I found therein a reference to the peptonizing of pollen by Urbach; this is reported in the *Annals of Allergy* for 1947. The same volume contains an obituary of Dr. Urbach, who died in 1946. The publication is therefore posthumous, which may explain the absence of publication of further exploration of the method. Urbach believed that the administration of peptonized specific foods by mouth was a specific for food urticaria. He called his products "propeptans", and proved by animal experiment that pollen propeptan had low toxicity but high immunological quality. He deduced from this that pollen propeptans should be of value in the control of hay fever, but did not mention any work to demonstrate this. He also presented analyses of the end products under varying conditions of digestion, which throw considerable light on certain difficulties in our own work and probably help to explain certain differences in the product we are using this season. It appears to be at least ten times as toxic and somewhat less effective. My present conclusion on that is that in striving after perfection by maintenance of pH we have taken away some of the virtue.

It seems that we have taken up where Urbach left off, that there is a great deal yet to be done, and that that great deal is work for a team of allergists and chemists who have ample clinical material at hand. Such conditions apply only in the United States of America, where chairs of allergy are not uncommon and hay fever lasts all the summer long; the work should thus return to its native land. Our work this year is designed to prove whether this method of treatment has enough merit to make it worth following up—no more, no less. If it should prove that it has such merit, then it is to be hoped that appropriate action will be taken by those who have the facilities.

[1] November 6, 1954.

AN INTERPRETATION OF THE RESULTS OF SKIN TESTING IN SEASONAL POLLINOSIS.

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Pollinosis may be defined as a state in which allergic symptoms (usually hay fever) are produced as a result of contact with pollens by a person hypersensitive thereto.

The diagnosis of pollinosis is based on an analysis of the clinical symptoms and the results obtained from skin testing with pollen extracts. In respect of the symptomatology, there is rarely any difficulty in establishing a diagnosis. The symptoms are found to occur at a particular period of the year when it is known that certain pollens are air-borne, and the time of onset and duration of the symptoms depend on the particular pollen, or pollens, to which the patient is clinically sensitive. The symptoms of which the patient complains are primarily due to the reactions produced by the pollens coming into contact with the mucous membranes of the eyes, nose, pharynx and bronchi. As a result, allergic rhinitis and asthma develop. Cutaneous manifestations may occasionally result from systemic absorption or direct pollen contact with the skin. This clinical diagnosis, based on the typical allergic seasonal history, can be corroborated, with rare exceptions, by the results obtained from skin testing with the pollens that are responsible for symptoms. A strongly positive reaction may indicate (and usually does) that the patient is clinically sensitive to the pollen in question, and will on proper exposure develop clinical symptoms. However, certain definite exceptions to this rule occur, and they must be taken into consideration when the results of the skin tests are being analysed.

A very definite fact which is noted on skin testing with the various grass species in patients complaining of seasonal pollinosis is the multiplicity of the positive reactions obtained. It is apparent that the average "sensitive" patient records a positive skin reaction in over 80% of cases to any of the grass pollens that are used for such testing. Since the best results in treatment depend upon desensitization with the specific allergens to which clinical sensitivity (that is, shock tissue sensitivity) has been acquired, the difficulty arises in deciding which of these many grass pollens that produce positive reactions are of importance, and as a consequence which of them should be included in treatment extracts. The actual number of individual pollen fractions that can be included in the one extract is limited for various practical reasons.

Therefore an analysis of the results of skin testing with pollens and an interpretation of the significance of such results seemed to be indicated. In this paper the subject matter is based on the results of skin testing of a consecutive group of patients whose symptomatology was purely or predominantly that of seasonal pollinosis, corresponding primarily to the grasses.

Method.

Fifty patients examined over the past eighteen months were selected for analysis as suffering from seasonal pollinosis on the basis of their symptomatology and clinical findings. The pollens that appeared to be mainly incriminated belonged to the Gramineæ (grass family). A routine general examination, as well as a more detailed allergic history-taking and examination, including radiography of the chest and nasal sinuses, was carried out in each case. Other special investigations were ordered where indicated. After these, diagnostic skin testings were carried out.

The diagnostic skin tests which were made on this series of patients were of two types: firstly, skin tests to determine pollen sensitivity; secondly, skin tests to determine sensitivity to other perennial inhalants, such as house dust, kapok or animal danders, or to ingestants such as foods, when the allergic history suggested an indication for such tests. For the purpose of this analysis the last-mentioned group of test reactions and their clinical evaluation are not included, although it is fully recognized that

in some cases of seasonal pollinosis both allergic and non-allergic causes other than pollen sensitization may be of contributory or even major importance in the production of clinical symptoms. In regard to the pollen tests, the following two groups were differentiated: (i) pollens of the Gramineæ (grass family)—50 patients tested; (ii) pollens of the other plant families—41 patients tested. The total number of pollen tests was 672. Of these, 419 were carried out with the grasses and 253 with the other plant families.

The dermal scratch technique was used in all cases as far as possible under a standardized procedure, the skin of the back being utilized on every occasion. Parallel vertical needle scratches, two inches apart and a quarter of an inch long, were made without drawing blood. A small amount of physiological saline was placed on each scratch, and to each was added a small uniform amount of the individual dried pollen extract which was mixed into a paste. A control test with saline only was made on each patient. Reactions were read usually after twenty minutes, when the maximum reactivity was reached. In cases in which more rapid and extensive reactions occurred, results were recorded much earlier and the testing material was removed. Such reactions should not be permitted to progress to their maximum, as not only do they give no further diagnostic information, but they may even proceed to generalized reactions. This did occur in two cases, with asthma, generalized urticaria and œdema of the periorbital tissues and of the larynx, mainly owing to the fact that rapid and extensive skin reactions were not noted at the time of testing. Adrenaline and antihistamines given by injection relieved the symptoms effectively. Less frequently reactions develop after a longer period, but still manifest the characteristics of an immediate direct reaction and are interpreted as such.

Now the characteristics of the immediate direct reaction are erythema and central weal formation, with or without pseudopodia. However, in the interpretation of the intensity of such reactions on skin testing, no uniform criteria exist amongst the various observers. Berkoff (1933) has suggested the use of a standardized scale; others use pictures of the reactions, in actual size, for permanent records. Both are too time-consuming for ordinary routine work. Probably the most satisfactory and uniformly adopted method is the "plus" system, a detailed description of which is given in an excellent discussion of skin testing by Vaughan and Black (1948). In this method, which has been used here, it is customary to record five possible types of immediate direct skin reactions. These are termed "0", "+", "++", "+++" and "++++" reaction (or "0", "1", "2", "3" and "4" reaction); "0" indicates no reaction when compared to a control test; "+" (or "1") indicates a slight but positive reaction; "++" (or "2") indicates a moderately strong positive reaction; "+++" (or "3") indicates a strongly positive reaction; "++++" (or "4") indicates a very strongly positive reaction. A "++" reaction, or above, therefore indicates definite and increasing skin sensitivity to the particular allergen being tested. However, for the present analysis, the reactions above were placed into one of the following three groups: group I signifies "0" reaction; group II signifies "+" reaction; group III signifies "++", "+++" or "++++" reaction.

Results.

It has been stated that the various plant species capable of causing seasonal pollinosis can be divided into the following two groups for the sake of simplification: (i) pollen-producing species belonging to the Gramineæ (grass family); (ii) pollen-producing species of other plant families.

In the southern area of Victoria the Gramineæ can be further divided into subfamilies, nine in number (Ewart, 1930), of which six are important as the main causes of seasonal pollinosis. In each of these six subfamilies, either two or four representative species of grasses have been selected for use in testing. That is to say, some 20 individual grasses are used, which it is believed cover adequately from a practical point of view the majority of those species capable of causing clinical symptoms.

Total Skin Reactions to the Grasses and Other Plant Families.

In Table I the results of the tests on all 50 patients are recorded for the grasses and for the other pollen-producing families.

Table I.

Results of the Total Number of Skin Reactions Obtained on Testing with (a) the Grass Pollens and (b) the Pollens of Other Plant Families.

Pollens.	Skin Reactions.			Total.
	Group I.	Group II.	Group III.	
Grass pollens	32 (8·0%)	40 (9·5%)	347 (82·5%)	419 (100%)
Pollens of other plant families	140 (55·3%)	37 (14·7%)	76 (30·0%)	253 (100%)

For the grasses, it is seen that in 8% of the total number of tests performed a negative result is produced. In 92% positive reactions occur, of which some 83% are moderately strong to very strong reactions. In the case of the other pollen-producing plants there is a considerable difference. Here 55% of the tests produced no skin reactions and only 45% positive reactions, and of these only 30% belong to group III (moderately strong to very strong skin reaction). It would seem, therefore, that in the southern area of Victoria considerable multiple sensitizations to the grass species occur, while in the case of the other plant families which produce allergenic pollen, though skin reactions do occur, they do so in a much smaller proportion of cases—namely, 45%.

Skin Reactions to the Subfamilies and Individual Species of the Grasses.

Under Table II the results of skin testing with the various grass pollens have been recorded in percentages—firstly for the different subfamilies of the grasses, and secondly for the individual representative species of these subfamilies.

When these results for the six main subfamilies are analysed, it is seen that the skin reactions produced by each individual subfamily of the grasses are practically identical with those obtained for the grasses as a whole (*vide* Table I). This is especially noted for the first three subfamilies (Festuceæ, Hordeæ, Aveneæ), for which 106, 98 and 75 individual skin tests respectively were performed. For the individual species it is again noted that the pattern of results is similar. In those species for which 25 or more individual tests were performed (nine species and an average of 36 tests), the reactions for groups I, II and III respectively are approximately 7%, 8% and 85%. Rye, Yorkshire fog, cocksfoot and Timothy grasses produced a slightly higher percentage of positive reactions. Wild oats, though quite common and a fair pollen-producer, produced the greatest percentage of negative results—some 16%. Of the remaining seven species, the numbers of individual tests for each are too small to permit any generalization to be made.

In summary, therefore, the results in this consecutive group of patients complaining of seasonal pollinosis and tested with the foregoing representative grass species, are as follows:

(*a*) The overall results show that 85% record a moderately strong to a very strong reaction (group III reaction); 8% record a positive but slight reaction (group II reaction); and 7% record no reaction (group I reaction).

(*b*) For each individual grass subfamily the results are approximately the same, the variation being small.

(*c*) For any individual species of grass from the first six subfamilies, the degree of skin reactivity recorded on testing is again similar to (*a*) and (*b*). The variation as expected is slightly greater, but not to a significant extent.

DISCUSSION: SIGNIFICANCE OF POSITIVE SKIN REACTIONS.

Since it is found that in the "average" patient with pollinosis, no less than 93% of the grasses tested produced positive cutaneous reactions, of which 85% were moderately strong to very strong, the question arises as to the significance of such results.

In allergic practice skin testing with various allergens is a routine procedure, and the accurate interpretation of such reactions as may thereby be obtained is of great importance in diagnosis and treatment. It cannot be too strongly stressed, however, that the results of skin testing give essentially confirmatory evidence only, and do not supplant a proper history and examination, both general and allergic, in each individual case.

TABLE II.

Results of the Skin Tests for (a) the Different Subfamilies and (b) the Individual Species of the Gramineæ.

Gramineæ.		Skin Reactions. (Percentage.)			Number of Individual Tests Performed.
Subfamily.	Species.[1]	Group I.	Group II.	Group III.	
Festuceæ ..	Sterile Brome	7·7	7·7	84·6	26
	Prairie ..	8·0	8·0	84·0	25
	Kentucky blue	0·0	15·4	84·6	13
	Cocksfoot ..	5·0	7·0	88·0	42
	Total ..	3·8	8·5	87·7	106
Hordeæ ..	Wheat ..	35·7	7·3	57·0	14
	Couch	6·0	0·0	94·0	17
	Barley ..	8·0	8·0	84·0	25
	Rye	2·4	7·1	90·5	42
	Total ..	9·6	5·8	84·6	98
Aveneæ ..	Wild oats ..	15·6	6·3	78·1	32
	Yorkshire fog	2·4	7·0	90·6	43
	Total ..	8·0	6·7	85·3	75
Agrostideæ ..	Timothy ..	3·0	5·9	91·1	34
	Bent	0·0	12·0	88·0	9
	Total ..	2·3	7·0	90·7	43
Phalarideæ ..	Sweet vernal	0·0	5·9	94·1	17
	Canary ..	11·8	11·8	76·4	34
	Total ..	7·9	9·8	82·3	51
Andropogonaceæ	Johnson ..	6·3	12·5	81·2	16
	Kangaroo ..	10·0	0·0	90·0	11
	Total ..	7·4	7·4	85·2	27
Miscellaneous	Paspalum ..	—	—	—	11
	Rhodes ..	—	—	—	4
	Maize	—	—	—	3
	Clover[2] ..	—	—	—	1
	Total ..	21·1	52·6	26·3	19

[1] The common names of the individual grass species and not their botanical names have been used here.

[2] Clover belongs to the family Leguminosæ.

In addition the visible criteria of positive immediate direct reactions, which are wealing and erythema, may be produced not only by the specific allergen-antibody reaction, but also by non-specific (that is, non-allergic) mechanisms. The trauma produced on testing, the glycerin or other preservatives used in test extracts, or high concentrations of the antigenic fraction alone, all may act as irritants with production of non-specific reactions. These reactions may be especially strong if there is a tendency to dermatographia. The use of controls in all cases and sufficient experience with testing are prerequisites in the differentiation of such specific and non-specific reactions from each other.

Allowance being made for the foregoing facts, it can be stated that positive immediate direct reactions indicate skin sensitization only to the allergenic fractions used. However, they do not necessarily indicate that the particular pollen so tested is responsible for the clinical symptoms complained of by the patient. This differentiation is important, since it is the rule rather than the exception that in many allergenic diseases multiple skin sensitizations occur. A positive reaction to a particular allergen means that there has been past exposure in a susceptible individual to the allergen in question with the production of skin-sensitizing antibodies. On dermal testing, the interaction of such allergen-antibody fractions probably liberates histamine or histamine-like substances, and these produce the typical weal and erythema of the immediate direct reaction. If at the same time sensitizing antibodies are formed in shock tissue, then on the subject's reexposure to the allergen clinical symptoms depending on the particular shock tissue involved may result. However, this generally held supposition is not fully supported by clinical and investigative facts; that the allergic mechanism involved in the production of acquired cutaneous sensitization is identical with the one concerned with acquired shock tissue sensitivity still remains to be proven. Irrespective of the mechanisms involved in either case, it must be fully realized that both sensitization of the skin and sensitization of shock tissue may occur together, or either may occur alone. In the latter case it is usual for skin sensitization to occur (at least clinically), rather than the reverse. In other words, the significance of a positive skin reaction to a particular allergen may be interpreted as follows: (*a*) The allergen has been the cause of past allergic symptoms, which have become latent even though skin sensitization has remained. (*b*) The allergen is the cause of present symptoms. (*c*) The allergen may, in the future, be the cause of symptoms due to the development of sensitization in shock tissue.

The patients reviewed in this analysis all showed positive reactions to pollen allergen on skin tests and clinical symptoms to pollen allergen from associated shock-tissue sensitization. However, each patient recorded positive skin reactions to many pollens and not just to one. Therefore the question arises, which of these pollens that produce positive skin reactions are also involved in shock-tissue sensitization and as a result produce the clinical symptoms complained of? This entails a consideration of certain basic criteria which have been formulated by Thommen, together with the problem of group and species specific sensitization to antigenic fractions.

Thommen's Criteria.

Thommen has listed the following five basic criteria which must be fulfilled before a pollen can be accepted as causing epidemic or endemic pollinosis: (i) The pollen must contain an excitant of hay fever or asthma. (ii) The pollen must be amenophilous (wind-borne) as far as its mode of pollination is concerned. (iii) The pollen must be produced in sufficient quantities. (iv) The pollen must be sufficiently buoyant to be widely dispersed. (v) The plant producing the pollen must be widely and abundantly distributed.

These criteria do not apply to sporadic cases of pollinosis, such as may occur in florists, botanists, gardeners, *et cetera*; under these circumstances the environmental factor is an artificial one, and as such would not apply to the ordinary case of seasonal pollinosis. The practical application of these criteria lies in the fact that of the hundreds of wind-pollinated plants, only relatively few fulfil all the points so listed. For example, Thommen (1931) found in a radius of one hundred miles of New York City some 270 individual grass species, yet only about five of these were found to be important causes of seasonal hay fever. Of course, in other localities the variations may be greater. However, the fact is that on the basis of such criteria the relative importance of pollen-producing plants in any one locality as causal factors of seasonal pollinosis can be evaluated. The majority of the representative grass species used for skin testing here apparently fulfil these criteria in relation to the southern area of Victoria. A few certainly do not; for example, Rhodes, Timothy, Johnson, and Kangaroo grasses are not of major importance, for they do not fulfil all the five criteria mentioned above. However, it is noted that these species still produce as high a percentage of positive skin reactions as do the other grass species (*vide* Table II).

Therefore the application of Thommen's criteria in an individual case of seasonal pollinosis is of value, in that those pollens which produce positive skin reactions can be differentiated into two basic groups. In the first group are those pollens that are of probable major importance in the production of shock tissue sensitivity reactions—that is to say, produce clinical symptoms—and in the second group are those pollens of minor or negligible importance. However, in the former group these criteria do not enable those pollens of possible major clinical importance to be differentiated much further. Therefore a consideration of the problem of group and species specific sensitization is indicated.

Group and Species Specific Sensitization.

Summary of Present Concepts.

It has long been recognized in seasonal pollinosis that patients record multiple positive skin reactions to pollen testing (*vide* Table I), and that there are no standard criteria for the selection of any particular grass pollens for use in treatment when this is based on the results of skin reactions alone. The problem of crossed immunological reactions between various related biological species is not new and has been the subject of much study, but the results obtained are often contradictory.

In reference to seasonal pollinosis, this problem can be simplified by considering that there are four possible types of antigenic fractions present in grass pollen which are capable of producing cutaneous and shock tissue sensitization. (*a*) A single common group antigen is present in the majority (or all) of the grass species. (*b*) Group antigens are present, each one of which is common to many individual species. (*c*) A species specific antigen is present for each individual species. (*d*) Combinations of these three types of antigens may occur.

Now it has been shown that many antigenic substances do contain a combination of different antigenic fractions; these fractions are (i) specific fractions for the individual substance alone, and (ii) fractions common to a group of related substances. For example, Tuft (1934) has shown that horse dander and horse serum contain a common antigenic fraction, and in addition horse dander contains an individual species specific antigenic fraction. The Salmonella group and serologically allied organisms contain common O-antigens, and H-antigens of specific and non-specific phases. The pneumococci contain both type and species specific antigens which are used for serological identification. Therefore, on this basis, it is a reasonable assumption that a closely identical biological family such as the Gramineæ would contain in their pollens common antigenic fractions and species specific fractions, and as a consequence multiple sensitizations could result.

Although this problem has not been clearly resolved, the bulk of experimental work on pollens definitely supports such a concept of group and species specific sensitivity, with the emphasis on the more dominant role of group sensitization reactions. The confirmatory work for this concept falls under the following two main headings: (i) the evidence from skin reactions of the immediate direct type obtained on testing with the different pollen species, and the results of treatment with one or several pollens in an extract on patients' skin sensitive to different pollens; (ii) the evidence obtained from indirect testing by the Prausnitz-Küstner method of inducing local passive sensitization of skin sites in non-allergic recipients with a sensitized serum from an allergic donor, and retesting such sites after a period of twenty-four to forty-eight hours with the various pollens. This method, of course, is time-consuming and requires good technique, and the variability of the reactions obtained must be realized.

Evidence from Direct Skin Testing and Treatment.—Thommen (1931), from the results of direct skin tests on some 30 patients, and using 81 grass species belonging to different genera, found that all such tests produced a positive reaction. He also noted that positive reactions occurred to the pollens of a grass species indigenous to Spain, to which the patients could never have suffered exposure. This means that common antigens between species must exist. Scheppegrell, in a comparable series of cases and using 100 different varieties of grass pollen, also found that all tests yielded a positive skin reaction. Chobot (1929) in treatment found that Bermuda grass pollen extract could desensitize the patient against Timothy pollen, and *vice versa*. Cooke and Vander Veer (1916) showed that persons sensitive to grass pollen almost without exception gave positive skin reactions to Timothy grass; they believe that a Timothy pollen extract alone is effective in the treatment of patients with grass hay fever. Similar confirmatory evidence has been obtained for the other plant families. However, other investigators, while fully admitting a high degree of crossed reactivity among the grasses, stress that more specific sensitivities also occur. Vaughan and Grubb (1938), in some 300 cases of grass sensitization, were unable to confirm that every grass species always produced a positive reaction to skin tests. Each individual grass cross reacted in approximately 65% of cases, the maximum cross reactivity being in the case of red top grass (84%). Similarly Vaughan, using 10 different species for skin testing, recorded that a positive reaction to only one species occurred in 17% of cases, and this could be to any one of the 10 species used; that is, a single species specific sensitization could occur. Watson and Kibler (1922) demonstrated that some of their patients sensitive to Bermuda grass yielded no reaction on skin testing with Timothy grass. Lamson and Miller (1927) were also unable to confirm the presence of complete multiple sensitizations in their grass-sensitive patients. Similarly in treatment the use of one or several standard grass pollens only for extracts does in some cases result in failure, owing apparently to the lack of certain specific pollen fractions. Henderson has shown that patients not relieved by Timothy grass extract often are relieved of symptoms when a mixed pollen extract is used containing those other pollens to which skin sensitivity is present and which fulfilled Thommen's criteria.

Evidence from Indirect Testing by Passive Induced Sensitization.—In 1921 Prausnitz and Küstner demonstrated the indirect method of skin testing. Here, if the serum of a donor suffering an atopic allergy and containing atopic reagins (specific antibodies) is injected intradermally into the skin of a normal non-atopic recipient, then the area so injected can be passively sensitized with the reagins, with the result that when such sites are tested with the atopens (specific allergens) to which the donor was sensitive, a direct immediate reaction results. This allergen-antibody reaction is a specific one. With this method of passive transfer, testing the problems of group and specific sensitization has been extensively investigated. Stull, Cooke and Barnard (1932), using sera from 11 grass-sensitive patients and seven individual grass species, found that each grass could desensitize the passively sensitized sites against any of the others, and therefore postulated that there was an active excitant (allergen) in all (these) grasses which was identical. Chobot (1929) states that Timothy and Bermuda grass pollens actively desensitize each other in passive transfer tests. Again, however, other investigators have found more evidence of individual group or species specific sensitization. For example, Coca and Grove (1925) found that Timothy grass desensitizes areas which had been passively sensitized to orchard grass, but that orchard grass did not always or completely desensitize against Timothy grass. Therefore they stated that allergens were present in Timothy grass pollen which were not present in orchard grass pollen. Rackemann and Wagner (1936), using Timothy, orchard or red top grass in the treatment of passively induced sensitized sites, found that a simultaneous desensitization to the other two usually resulted; but the important fact was that no single one of these three grasses always and invariably produced such desensitization to the others. It is noted incidentally that Timothy, Bermuda, orchard and red top grasses, with which much experimental work has been done, show a high degree of group antigenicity; yet they belong to separate subfamilies of the Gramineæ, on a morphological classification.

Comment.—While one acknowledges the fact that the whole question of biological antigenicity is still not fully clarified, a summary of the foregoing investigations of

group and species specific sensitivity within the Gramineæ can be stated as follows:

1. Group antigens are present to a considerable degree between the different species, and account for the multiplicity of positive skin reactions found on testing.

2. The exact types of such antigens are undetermined, but the consensus of opinion supports the view that they are (*a*) common group antigens to the majority of the species, and/or (*b*) group antigens, each common to several unrelated or related species.

3. Individual species specific antigens may occur, but rarely if ever do they produce cutaneous sensitization alone. However, this does not mean that they cannot be the "dominant" antigen, and as a result the major or only cause of shock tissue sensitization and therefore of clinical symptoms.

4. Any of these antigens are capable of producing sensitization of the skin and/or shock tissues.

Comparison with Results in this Series.

While the foregoing statements were accepted, the 50 patients were subjected to a further analysis to determine how they would conform to the problem of group and species specific sensitivity. Under Table III the patients were divided into two groups: (i) those who gave a positive skin reaction to all grass pollens tested; (ii) those who failed to react to one or more tests, their reactions to the remaining tests being positive. The variation of the intensity of the skin reactions was also recorded.

TABLE III.

Results of the Skin Reactions to the Grass Pollens.

Skin Reactions.	Number of Patients.	Number of Pollen Tests.[1]	Intensity.		
			Group I.	Group II.	Group III.
Positive to all pollens tested	36	271	0	19	252
No reaction to one or more; positive reactions to the remainder of the pollens tested	14	129	28	11	90
Total.. ..	50	400	28	30	342

[1] These results are taken from the first six subfamilies of the Gramineæ, with which some 400 out of a total of 419 grass pollen tests were done (Table II).

From these results the following conclusions have been drawn.

1. In no case did a single positive skin reaction alone occur, which means that a single species specific sensitization was not found in this series of 50 cases. This finding is in agreement with the results of Thommen, Scheppegrell and others, but is contrary to those of Vaughan and Grubb, who quote single specific sensitization present in some 17% of their cases. The minimum degree of skin reactivity occurred in Case XIII, in which six grass pollens tested produced respectively reactions "0", "+", "+", "0", "++++", "+". In all other cases an increasingly higher degree of positive reactivity was recorded. The conclusion is therefore that single species specific sensitization (cutaneous) does not occur.

2. It was also noted that only 36 patients yielded a positive skin reaction to every pollen tested, the remaining 14 patients recording a negative response to one or more tests, the remainder being positive, and these negative responses could be to any one of the pollens used. The conclusion is therefore that a single group antigen common to all (these) species did not occur.

3. The actual intensity of the positive skin reactions varied. This fact is one which is recorded by all investigators, irrespective of their views on the predominance of common group antigens and/or species specific antigens. In Table III there were 372 positive reactions, equal to 93% of the total skin tests performed, and these in detail consisted of a "+" reaction in 7·5%, a "++" reaction in 12·2%, a "+++" reaction in 18·8% and a "++++" reaction in 61·5%. If one antigen common to the various species only is present, then all skin reactions to their pollens should be of similar intensity, since the two factors concerned in the skin reactions in the individual patient—namely, the skin sensitizing antibodies and their corresponding pollen antigens—should be the same. (The obvious objection to this statement would be that identical amounts of different pollens contain varying amounts of group antigen, and thereby produce variable reactions. This cannot be true, since reactions of species vary unpredictably in intensity in relation to each other. For example, if the reactions of the two grass species prairie and sterile Brome are compared, and it is assumed that the latter species contains more common group antigen per unit volume, then its reactions on testing should consistently be of greater intensity in different patients. However, this is not so; a "+" to "++++" reaction can occur for each grass pollen in any combination. Similar results are recorded when any other number of species are compared.) The fact that such reactions are of different intensities with a greater incidence of strongly positive to severe reactions indicates that a single common antigen cannot be present, and that there must be other antigenic fractions with their corresponding antibodies which produce the reactions. The conclusion is therefore that antigenic fractions common to several species—that is, common group antigens—are present.

This concept of antigenic fractions common to different species leads to a consideration of their actual distribution among the Gramineæ as a whole. It would be expected that the more closely related the individual species were to each other, then the more common would be group antigens to them. Therefore in any case of the multiple sensitization type, in which individual species of the same morphological group (that is, subfamily) are tested, they should produce a greater incidence of positive skin reactions than would occur when in the same case individual species from different subfamilies were tested and both results were compared. In Table IV are recorded the results of those tests in which two or more species from (*a*) the same subfamily and (*b*) different subfamilies were tested in the individual patient. In the former case there were 123 instances of such group testings, in the latter 409.

These results would indicate that there is little difference in the incidence of positive skin reactions when the grass species tested, either from the same subfamily or from different subfamilies, are compared in each case. Therefore group antigens common to the species of individual subfamilies are not present to any greater extent than are those between "unrelated" species of different subfamilies. Of their actual distribution among the Gramineæ very little can be stated, except that they apparently occur in an irregular and unpredictable manner.

The important fact which must be realized, however, is that the Gramineæ have been systematically classified into the various subfamilies, genera and species on a predominantly morphological basis, and not on the possible antigenic groupings which exist between the various species. Investigation of this latter relationship would seem to be of little value in the botanical field, but would be of clinical importance to allergists. If a classification on the basis of antigenic relationships could be made, then it might well be entirely different from the present one so universally adopted, and the results of pollen skin tests would have a more regular and predictable pattern.

Identical or closely related antigenic groupings in living cells predominantly occur in those cells which have a close systematic relationship. Similar related antigens may, of course, occur in cells without such a relationship; for example, Forsmann's heterophile antigen is present in bacteria, red blood cells, and certain tissue cells. Pneumococcus type I and certain coliform bacilli are serologically related. However, these are the rare exceptions and do not alter the general rule of systematic relationship between antigens. That antigens may be specific in a

systematic sense depends on the manner in which chemical substances or the enzyme systems that synthesize them are distributed in Nature so as to form these specific antigens, and this distribution may have diverged in an entirely different manner from the morphological variations that have arisen in the development of new species. In this way common antigenicity between species and morphological characteristics may have become widely separated among the grasses.

TABLE IV.

Source of Species.	Number of Group Tests.	Results of Skin Tests.	
		Positive to All Species in Each Test.	Positive to Some Species, Negative to Other Species in Each Test.
Same subfamily ..	123	106 (86·2%)	16 (13·8%)
Different subfamilies	409	341 (83·4%)	68 (16·6%)

The conclusion is therefore reached that group antigens common to individual species of the Gramineæ occur in an irregular and unpredictable manner. This may be due to the fact that the species are grouped into genera and subfamilies on a morphological basis, and not on their antigenic relationship.

Shock Tissue Sensitization.

On the basis of the conclusions which have been formulated from this analysis, and with due consideration of the currently held concepts of other investigators as presented above, it can be stated that on skin testing with different pollen species in cases of seasonal pollinosis, multiple positive cutaneous reactions are the rule. These reactions are due to the sensitization of the skin tissues to group antigens common to different individual species in a complex and unpredictable pattern, and in addition there is present sensitization to species specific antigens. This species specific sensitization is not found alone, at least in reference to a single cutaneous sensitivity, but is always associated with other demonstrable antigenic sensitizations.

However, when the problem of shock tissue sensitization is considered, although there can be little doubt on clinical and experimental grounds that the pollen antigens responsible for the multiple cutaneous reactions are also involved in shock tissue sensitivity, and on exposure to these tissues are the cause of clinical symptoms, two important facts must be noted. Firstly, the allergic mechanism involved in each case may not be the same, and certain clinical and experimental observations do suggest such a dissimilarity. Secondly, and of greater importance from a practical point of view, the sensitivity reactions occurring in shock tissue are possibly due to one or at the most a small number of pollen antigens, which antigens may be common group and/or species specific type. This supports Vaughan's concept of the occurrence of single species specific sensitivity—present in 17% of his cases—but only in reference to shock tissue and not to cutaneous involvement, which in all cases recorded in this series is of the multiple sensitization type.

Wodehouse (1949) believed in this respect that patients with pollen sensitivity of the multiple sensitization types have in general one major sensitization upon which the other sensitizations depend regardless of their number. The structure of pollen antigens is a mosaic one, in which there is a major antigenic fraction which is group or species specific and may be shared, if at all, by phylogenetically related species; in addition there are minor antigenic fractions which are common to related and unrelated species in an irregular and unpredictable manner; all these are capable of producing clinical symptoms. There is confirmatory evidence for this concept of a major shock tissue sensitization to one or a few closely related species. Cooke and Vander Veer (1916) showed that Timothy pollen invariably produced a positive reaction on skin testing, but more important still, that an extract of this pollen alone in treatment produced an adequate clinical response in the majority of their cases of grass pollinosis, despite the fact that multiple skin sensitivities to other pollens were present. This could not be due to the group antigens common to Timothy and the other species, because the use of extracts of other species did not produce an adequate clinical response if Timothy pollen was not included. This indicates that a more specific and "dominant" antigenic fraction is contained in the Timothy pollen, which is involved in the shock tissue sensitization reactions.

Other investigators in other localities found a similar tendency for one or more pollens to predominate as the major cause of symptoms. In actual fact in America some three species, Timothy, June and Bermuda grass, are the predominant cause of clinical symptoms of grass pollinosis, accounting for some 90% of cases. That in a minority of cases the response to treatment with a single pollen extract is unsatisfactory does not discount the importance of major clinical sensitivity. It is believed in these cases that the major sensitivity is shared by more than one pollen antigenic fraction, and this accounts for the satisfactory clinical response that occurs when a mixed extract is used. It must also be realized that some patients, irrespective of what type of extract is used, will give a poor response to any method of pollen desensitization.

In reference to the southern area of Victoria, the importance of certain species as the predominant causes of clinical symptoms is also apparent. The time of seasonal pollinosis due to the grasses extends from early September to January, the maximal incidence and severity of symptoms occurring from November to December, a period of some six to eight weeks. The species whose flowering times correspond to this period of maximal symptoms, and which obviously fulfil Thommen's criteria as well as producing the best results in treatment for the majority of patients, are the rye grasses—perennial rye (*Lolium perenne*), Wimmera rye (*Lolium rigidum*), and Italian rye (*Lolium multiflorum*)—cocksfoot or orchard grass (*Dactylis glomerata*), and Yorkshire fog or velvet grass (*Holchus lanatus*). Other species are important as causal factors when symptoms are severe at an earlier or later period than the usual one from November to December. For example, in September and October, sterile Brome (*Bromus sterilis*), prairie grass (*Bromus unioloides*), June grass (*Poa pratensis*), and in January and February *Paspalum dilatatum*, are common causes of pollinosis. However, these do not dominate the clinical picture, in comparison with rye, cocksfoot and Yorkshire fog grasses, which seem to be the species containing the dominant antigens as regards clinical—that is, shock tissue—sensitization.

On analysis of the results of skin testing with these three grasses of major clinical importance, it was noted that the percentages of positive reactions for rye grass, cocksfoot and Yorkshire fog grasses were 97, 94 and 97 respectively—a very high degree of positive reactivity, especially when allowance is made for error in recording a response as negative. Also in 45 cases of the total 50, either two or all of these three grasses were tested, and in detail these results were as follows. In 32 cases the three grasses were tested on the one patient; in 29 of these cases all three produced positive reactions, and in three cases only did one of the grasses produce a negative response, the other two results being positive. In the remaining 13 cases two grasses were tested on the same patient, and both produced positive reactions in every case. This gives the very high proportion of 42 cases out of 45 in which two or three grasses when tested on the same patient produced positive reactions. Now rye, cocksfoot and Yorkshire fog grasses belong to different subfamilies of the Gramineæ—namely, subfamilies Hordeæ, Festuceæ and Aveneæ respectively. The above-mentioned high incidence of positive reactivity to their pollens in the same patient would tend to support one of the conclusions reached earlier—namely, that antigenic relationship between species could possibly produce a different classification from the present one based on morphological characteristics. Further investigative work between their common and specific antigenic

relationships is indicated, especially as they are of such clinical importance and produce such a high incidence of positive reactions when tested together on the same patient.

The conclusion, then, is reached that shock tissue sensitization and consequently the production of clinical symptoms appear to be due in the majority of cases to a limited number of antigenic fractions only. Though exceptions to this do occur, owing to antigenic fractions of other species being the dominant sensitizers of the shock tissues, they apparently do so in the minority of cases only.

SUMMARY.

1. A brief account is given of the methods used for skin testing in seasonal pollinosis and of the interpretation of the reactions obtained thereby.

2. The results of cutaneous testing with pollens in a consecutive series of 50 cases of uncomplicated seasonal pollinosis are recorded.

3. These results show a very high incidence of positive skin reactions (92%) to such tests with the Gramineæ (grass family).

4. The significance of such a high incidence of positive reactions is discussed, with special reference to (*a*) Thommen's criteria for the selection of individual pollen species as possible causes of clinical pollinosis, and (*b*) the importance of group and species specific sensitization among the pollens and their application to the results of skin tests obtained in this series of cases.

5. The relationship between shock tissue sensitization and cutaneous sensitization with the same pollen antigenic fractions is considered.

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Reports of Cases.

AN EPIC IN NARCOLEPSY.

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THERE can be few tales that would justify the title of an epic. This story concerns a man, H.C., aged twenty-six years, a narcoleptic. I first examined him in 1937, and at that time he was complaining of symptoms from which he had suffered for the preceding two years, and which were obviously due to narcolepsy. These attacks of pathological sleep usually commenced at about ten o'clock in the morning and recurred at intervals during the day. They were so insistent and of such intensity that, despite the fact that it meant a lower status and a lower salary, he was compelled to apply for a transfer to another department of the firm in order to conceal his disability.

This request for transfer mystified his employers, as he was extremely capable and energetic, showed great promise, and was obviously in line for high executive office. However, his transfer gave him greater freedom of movement and enabled him to find quiet corners where he could sit down for the necessary three or four minutes when his pathological sleep turns overcame him; for him the lavatory became a dormitory. He was also relieved from sitting in the office of senior members of the firm, thereby avoiding embarrassment, as he was inclined to talk confused rubbish or write unintelligible nonsense in the preliminary stages of going off to sleep.

He cultivated an amazing technique for covering up his symptoms, and became quite adept at resuming conversations which had been broken off by his short and compulsory sleeps.

As is usual with narcoleptics, he was also subject to cataleptic attacks. Among other things, H.C. was a first-rate athlete, but he had to give up all sport because of these cataleptic attacks. For instance, he was an interstate hockey player, but was forced to resign because, if he became excited or tense in a match, he would drop toneless to the ground as if he had been poleaxed. He would do the same thing while waiting to smash a lob at tennis, and he would collapse if much depended on his putt at golf. He dared not laugh, and on more than one occasion at the theatre he paid exaggerated tribute to the comedian by sliding out of his seat into the aisle. He was enormously self-conscious about his symptoms and went to extraordinary lengths to disguise them.

When I first examined him I prescribed half-grain doses of ephedrine. He improved dramatically and completely lost his symptoms. So absurd did this seem to him, and so convinced was he that it must be coincidence, that on several occasions during his first few weeks of treatment he gave up the ephedrine, only to find his symptoms return. On one or two occasions I tried him on "Benzedrine" or "Luminal", but neither drug was nearly so effective as ephedrine, and he has remained on ephedrine ever since.

During the next few years I saw him occasionally. Then, one day in May, 1940, he turned up in an Australian Imperial Force uniform. I thought he must be masquerading, but he was deadly serious. I pointed out to him how ridiculous it was for him to think that he could serve, much less go on active service. He replied that he was going anyway, however absurd it was, and that as a matter of fact his unit was leaving for overseas almost immediately. I repeated that it was impossible and that I would ring Victoria Barracks to inform the authorities about him. His reply to this was that I would be wasting my time, that I could not stop him, and that if I did inform on him he would merely go to another State and re-enlist under another name. He said that he had come to see me only to obtain a prescription for 5000 ephedrine tablets and a spare prescription for the same quantity in case of accident; and he led me to believe that he considered my reception of him rather poor.

He was quite determined; I was convinced I could not stop him; and so, rightly or wrongly, I gave him the prescriptions. I must confess that I was intrigued, but I really thought that his symptoms would find him out. They did; but not before he had done four years of strenuous active service.

I heard nothing of him until about June, 1944, when I received a telephone call from Major Moss, from one of the New South Wales military hospitals in the country. He told me that a man under his care, H.C. by name, had told him an incredible story about army service, narcolepsy and ephedrine. I assured Major Moss that his story was true, and we both agreed that he should be "boarded out".

Subsequently an equally incredulous officer of the Medical Discharge Board, the late Edgar Stephen, telephoned me about this man. I asked Colonel Stephen if he would be good enough to ask H.C. to come and see me when he received his discharge.

H.C. duly came to see me and told me his story. When I asked him to put it on paper he protested, but finally gave in, and his story in his own words is printed below.

In a covering letter this remarkable man apologized for his delay in sending it to me, but explained that he had had to rewrite it several times to avoid unnecessary detail.

I enlisted in the A.I.F. as a Private in an Infantry Battalion in May, 1940. I left with my unit for overseas in October of the same year, taking with me five thousand half-grain tablets of Ephedrine (in bottles of 500) which represented approximately 18 months supply. The daily dose consisted of 10 half-grain tablets taken at the rate of four, three and three at about four hour intervals.

After arriving in the Middle East, the next few months were spent between Palestine and Egypt until February, 1941, when I travelled with my battalion to Libya in North Africa to take part in our first operation.

Before leaving Palestine, I placed 1,000 half-grain tablets of Ephedrine in my kit bag, before it was sent to the Kit Store in Alexandria for storage and for safe keeping. I took this precaution to establish a reserve and to avoid losing my entire stocks, should anything disastrous happen to those which I carried on me, which was a possibility under the circumstances. However, the balance I carried with me, which were sufficient to last me for about nine months.

My first action was a "Strategic Withdrawal" which was done in stages over a distance of about 300 miles, occupying a period of over three weeks, and eventually terminating in Tobruk, which developed into what was later known as "The Siege of Tobruk". At the end of the seven months which followed we were relieved by British troops and evacuated from Tobruk in a destroyer to Alexandria, then to Palestine, where I was very pleased to see my kit bag containing the thousand tablets, as my supplies by this time were particularly low.

During the latter part of the Tobruk campaign I was promoted to a Warrant Officer Class II and appointed Company Sergeant-Major, and shortly after my arrival back in Palestine I was sent to do a six weeks training course at a Middle East School that was noted for its severity and high standard of Efficiency. It certainly justified its reputation, and although I didn't excel, I managed to obtain a pass. This no doubt sounds unimportant, but to me, under the circumstances, was rather a severe test, and the fact that I successfully completed the course without incident provided me with considerable satisfaction.

While on leave in Jerusalem I purchased 5,000 half-grain tablets of Ephedrine to replenish my depleted stocks. These were very poor quality in comparison with the Australian product, as they were very large and deteriorated very quickly. However, although quite a lot were lost they served a useful purpose under conditions which existed while out of action. I still had the balance of my original supply together with several small quantities that I had received since arriving back in Palestine, which were sent from Australia in response to my requests during latter stages of Tobruk when the Ephedrine position looked rather grim, but fortunately were not required. These I kept aside for use in the next action, which happened to be the "Battle of El-Alamein", a very grim affair which lasted twelve days and I consider myself very lucky to have survived.

I returned to Australia in February, 1943, and after a period of leave travelled with my Unit to North Queensland where I underwent four months jungle training in preparation for future operations in New Guinea.

I embarked for New Guinea in July, 1943, and took with me on this occasion 5,000 half-grain tablets of Ephedrine which I had purchased since returning to Australia. Following a few weeks practical exercises, preparation, etc., at Milne Bay, I embarked on Landing Craft for the amphibious landing for the attack on Lae, of which my battalion was the spear head.

Before leaving Milne Bay I placed 2,500 tablets of Ephedrine in my pack, containing change of clothing, spare boots, etc., which was supposed to follow with other stores after the fall of Lae, or when the situation permitted. On these operations we carried only essential battle equipment, the bulk of the load comprising rations carried in the haversack and ammunition in the ammunition pouches. In order to make the extra space to carry the Ephedrine I had two extra pouches attached to my haversack in which I carried sufficient for about nine months. Owing to a change of plan, the packs were not forwarded on, but held at Milne Bay and later sent to the mainland, so I was obliged to carry on with the tablets I carried with me.

Closely following the fall of Lae a further amphibious landing was made for the attack on Finschafen, after which the fighting continued in the mountainous country followed by the fall of Sadelberg and subsequent pursuit of the Japs along the coast ending up at Cio.

I returned with my battalion to Australia in March, 1944, reaching the mainland with only sufficient tablets to last about a week, which was more good luck than good management.

At the termination of my leave, I purchased a further 4,800 half-grain tablets of Ephedrine in anticipation of further campaigns up North, and it was on my way to rejoin my battalion in North Queensland that I had an attack of malaria at Brisbane. It was about mid-day and I intended taking the Ephedrine after I had seen the Medical Officer. However, the malaria condition appeared to increase rather suddenly and I apparently forgot to take the ephedrine. Spasmodically I was completely helpless and only able to walk a few steps at a time with assistance, and under extreme difficulty. While recovering in hospital at Tamworth I decided that I would be carrying too big a handicap with addition of malaria, and I would be serving no useful purpose by continuing under the circumstances, particularly in view of the strenuous campaigns to follow. I had had over four years' service, so I discussed the matter with a Senior Medical Officer at the hospital for the first time and I was subsequently discharged in July, 1944.

With the exception of the Ephedrine which I purchased in Palestine I used only Burroughs Wellcome "Tabloid" brand ephedrine tablets throughout my service. The supply which I took with me to New Guinea were particularly well packed in bottles of 100 and it was due to this and their quality that I attribute the success with which I was able to carry them, under the appalling conditions, through the New Guinea campaign.

I consider my medical record was particularly good, while on service. Shortly after enlistment while in camp at Ingleburn, I had an attack of boils on both legs which cleared up after a few weeks hospital treatment. Throughout my service in the Middle East I enjoyed the best of health. During the New Guinea campaign on one occasion I received treatment at a Main Dressing Station for acute bronchitis and an infection of both ears. After a few days rest, however, I was able to return to my unit. The bronchitis cleared up, but the ear trouble which was a common complaint among the troops continued until I left New Guinea. It was not until just prior to discharge that I received hospital treatment for malaria, which I mentioned previously. Since returning to my civil occupation I have found it necessary to increase my daily dose of ephedrine to 15 half-grain tablets per day.

SUBCLAVIAN ATHEROSCLEROSIS, CORONARY ATHEROMA AND ANGINA PECTORIS WITHOUT ELECTROCARDIOGRAPHIC CHANGES.

By A. J. BARNETT and J. R. E. FRASER,
Clinical Research Unit, Alfred Hospital, Melbourne.

ATHEROSCLEROSIS, which may produce diverse symptoms, usually affects the cerebral, coronary or limb vessels predominantly, and diagnosis of the lesion is not difficult. Occasionally the manifestations are atypical or their combination is unusual, so that even this common disease may present diagnostic problems. With the wealth of modern investigational methods, it might be expected that these would be readily solved. However, in the following instance the disease baffled the investigators; it is reported

with the hope that future investigators may be more successful.

Clinical Record.

A man, aged forty-four years, was admitted to hospital for investigation of recurrent pain in the chest. He had first noticed this while doing respirator drill in the army some ten years previously, and it had become worse four years prior to examination after an attack of appendicitis, and had been particularly severe in the past two years. It was produced by exertion, particularly walking up-hill, and consisted of a gripping pain across his chest going down the left arm to the wrist and the right arm to the elbow. It caused him to stop his activity, after which it subsided in five to thirty minutes. In spite of this symptom, he had continued with heavy physical work until a few days before his admission to hospital. He also suffered from attacks of pain while at rest. These had usually occurred in the early evening or the small hours of the morning; they had lasted up to two and a half hours, and injections of morphine had been required for their relief. During the past year he had been slightly short of breath on exertion and had suffered from attacks of shortness of breath at night — usually about 1 a.m. For the past ten years he had also suffered from pain in his right leg, most severe in the calf, but extending from the hip to the ankle. This was produced by walking, and occurred synchronously with pain in his chest; it was relieved by five minutes' rest. For the same period he had noticed a "cramping sensation" in his left arm if he held it above his head for more than a very short time. He had also suffered for the past sixteen years from epigastric discomfort after meals, relieved by antacid powders. Apart from this and an attack of appendicitis four years before his admission to hospital, he had not suffered from any serious illnesses. During the course of his illness the patient had consulted several physicians, one of whom has kindly given us a copy of his notes made about five years prior to the patient's admission to hospital. These indicate that the patient was normotensive, that cardioscopic examination revealed no abnormality, that electrocardiograms (including chest leads) were normal (Figures I and II), but that his left brachial and radial pulse could not be detected. A clinical diagnosis of *angina pectoris* had been made.

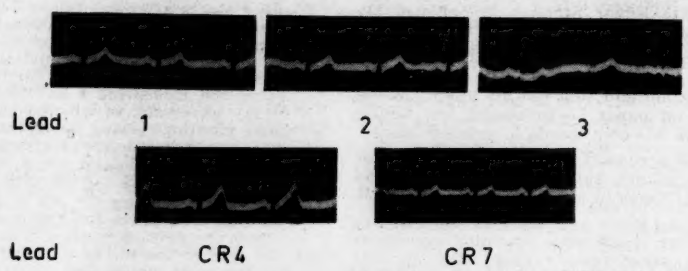

FIGURE I.
Electrocardiographic tracings approximately five years prior to the patient's admission to hospital.

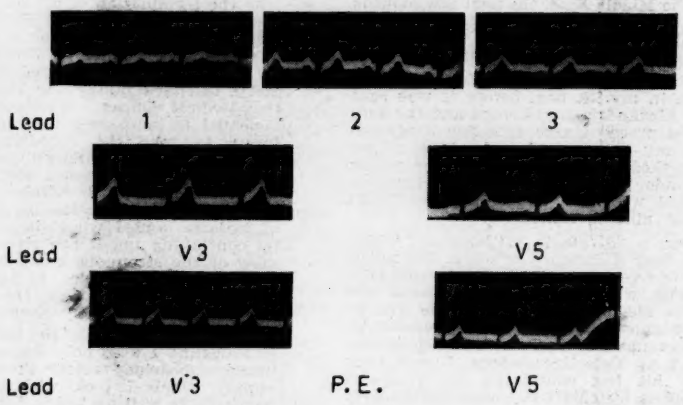

FIGURE II.
Electrocardiographic tracings approximately one year prior to the patient's admission to hospital. "P.E.", post-exertion.

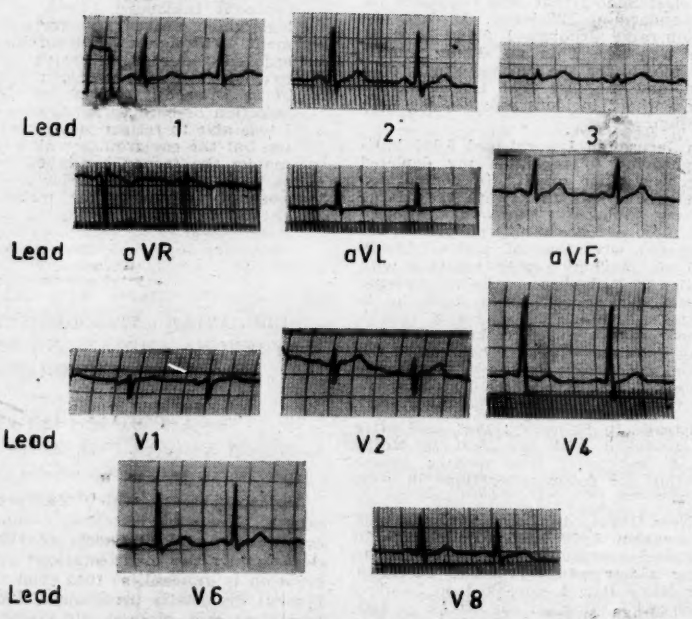

FIGURE III.
Electrocardiographic tracings after the patient's admission to hospital; retouched for clarity.

On examination, the patient was seen to be a thickset, muscular man. The blood pressure in his right arm was 130 millimetres of mercury, systolic, and 78 millimetres, diastolic, and in his left arm 80 millimetres of mercury, systolic, and 65 millimetres, diastolic. His chest was voluminous and the expansion was poor. The cardiac impulse could not be felt, but the heart sounds were normal and there were no bruits. The pulses in his right arm were normal, but the left brachial pulse was weak and the left radial pulse was almost imperceptible. His hands

were normal in appearance, but when they were repetitively clenched above his head the left palm became abnormally pale. The femoral pulses were normal, but the popliteal and ankle pulses were slightly weak. There were no other signs of arterial insufficiency in the lower limbs.

The step test gave the following results. Pain in the chest and right calf occurred on walking an average of eight times over the steps (two steps, each nine inches high).[1] The pain was relieved by one to two and a half minutes' rest. In the grip test, pain in the first left dorsal interosseous muscle and forearm was produced by 18 to 31 "grips" (squeezing a length of rubber hose connected to a sphygmomanometer to produce a pressure of 200 millimetres of mercury every five seconds).

Radiographic and cardioscopic examination showed no abnormality of the heart or great vessels and no bony abnormality in the neck or thoracic outlet region.

Scalar electrocardiography (including the use of standard leads, unipolar limb leads and chest leads) revealed no abnormality (Figure I). After exercise to the extent of producing chest pain, the tracings were still normal.

Plethysmography gave the following findings. The resting blood flow in each foot was normal (right, 2·4 millilitres per 100 millilitres per minute, left, 1·8 millilitres per 100 millilitres per minute), there being a normal increase from an arterial occlusion reactive hyperæmia test (right, 7·9 millilitres per 100 millilitres per minute, left, 8·3 millilitres per 100 millilitres per minute). The time taken to attain the maximum blood flow was slightly prolonged (right one and a quarter minutes, left one minute, after release of arterial occlusion).

Calorimetry showed that the resting heat elimination of his left hand was 32·4 calories per 100 millilitres per minute. There was no increase from a "reflex" hyperæmia test, consisting of immersing the other hand and forearm in water at 44° C.

Arteriography of the arterial tree of the left arm was unsuccessfully attempted.

Intraarterial pressure tracings showed no significant differences in the pressures in the right brachial artery and left femoral artery. The pressure-pulse tracings were normal in contour.

A left cervico-dorsal sympathetic blockade was produced by the injection of procaine hydrochloride followed by a long-acting anæsthetic agent ("Efocaine"—Crookes Laboratories) in the region of the upper dorsal sympathetic ganglia. This produced a left Horner's syndrome and warming of his left hand which lasted for several weeks. However, there was no improvement in the responses to the grip test or the step test. He still suffered from spontaneous attacks of chest pain, unmodified in extent or severity.

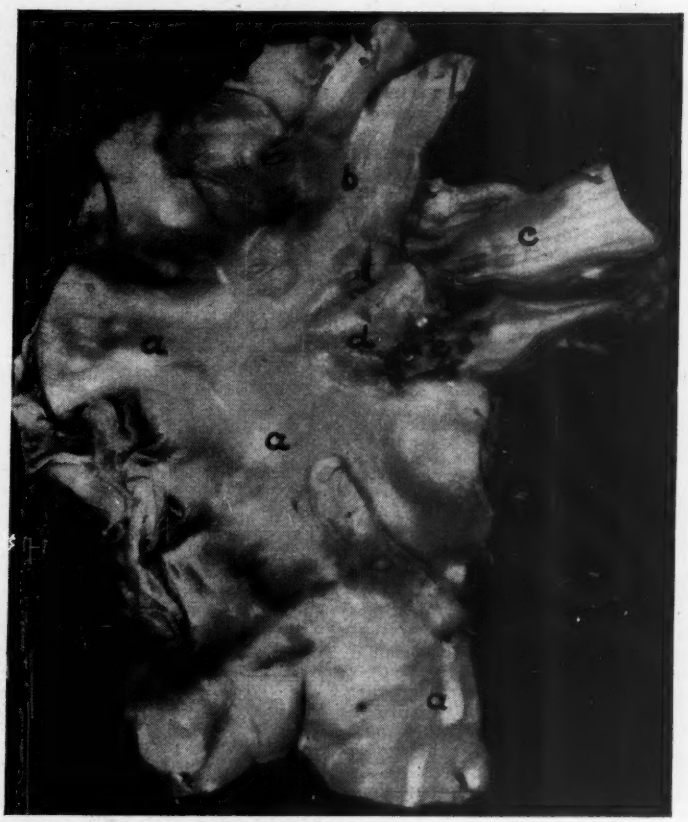

FIGURE IV.

Specimen of arch of aorta and its branches, opened out: (*a*) atheromatous plaques in aorta; (*b*) innominate artery; (*c*) left common carotid artery; (*d*) calcified and ulcerated atheromatous plaque at the origin of the left subclavian artery; (*e*) organized thrombus in the lumen of the left subclavian artery. Note: the apparent narrowing of the origin of the left common carotid artery is an artefact due to difficulty in pinning out the formalin-fixed specimen.

The patient was discharged from hospital with the diagnosis still uncertain in spite of exhaustive investigation. He continued to have frequent attacks of chest pain, requiring injections of morphine from his local physicians, who suspected that he was becoming addicted to this drug. The very weak left radial pulse and low blood pressure in the left arm were the only definitely abnormal physical signs. It was considered that there might be some lesion in the left thoracic outlet region obstructing the subclavian artery, and also causing pressure on nerves and so producing pain in the chest simulating *angina pectoris* (Reid, 1938). For this reason, and because the patient's life was one of extreme misery because of frequent attacks of severe pain, he was readmitted to hospital for surgical exploration.

Under general anæsthesia (thiopentone, nitrous oxide and oxygen), Dr. H. C. Trumble exposed the left thoracic outlet region. The subclavian artery was small and the pulsation in it was poor. No obstruction to the artery was discovered even when it was traced back to the origin of the vertebral artery. After completion of the operation, the patient's pulse became imperceptible and his blood pressure unrecordable; he died within a few minutes.

Necropsy Findings.

At necropsy, the significant findings were limited to the heart and great vessels. The heart weighed 18 ounces (540 grammes). The myocardium was very fibrotic, and there was an old infarct in the anterior part of the septum. Both coronary arteries were grossly atheromatous, so that in places no lumen could be demonstrated. The aorta was also grossly atheromatous. A calcified plaque was situated

[1] These steps are similar to those used by Master and Oppenheimer (1929) in their "exercise tolerance test".

at the origin of the left subclavian artery; organized thrombus was adherent at this point and projected into the lumen of the subclavian artery. The plaque and attached thrombus practically occluded the origin of the artery (Figure II). Microscopic examination of the aorta showed "atheroma only; no evidence of syphilis".

Discussion.

In view of the necropsy findings, the cause of this man's symptoms is clear: the chest pain was due to cardiac ischæmia from coronary atheroma; the cramp in the left arm and poor radial pulse were due to the occlusion of the origin of the left subclavian artery from atheroma. Although the arteries of the legs were not dissected, it is probable that vessels to calf muscles were also obstructed by the same disease.

The patient's history merits consideration from three aspects. First, it serves to emphasize that atheroma may cause widespread, apparently unrelated symptoms. Some of these, such as *angina pectoris* and intermittent claudication, are well recognized. However, a suitably situated atheromatous plaque, particularly if complicated by thrombosis, may block any artery. Occlusion of the large arteries of the upper limb from this cause is rare, although its occurrence at the origin of a subclavian artery is recognized (Allen, Barker and Hines, 1946).

Secondly, it demonstrates very strikingly the limitations of special tests used in the diagnosis of *angina pectoris*. It is well known that radiographic examination of the heart may show no abnormality (White, 1951), but electrocardiography is usually regarded as a very reliable diagnostic test. Thus White (1951) makes the following statement:

> *Electrocardiograms are of the greatest importance.* They are often normal with slighter degrees of coronary heart disease, especially between (not during) attacks of angina pectoris, but in advanced coronary heart disease they usually show changes in the Q and T waves and often in the S-T segments . . . .

Wood (1950) believes that the taking of an electrocardiogram after effort is very important, but states that even this may remain normal in a few cases of true angina. Pordy and his co-workers (Pordy *et alii*, 1952) believed that it could be practically excluded if "two-step" test responses were normal. However, it is surprising that coronary disease as gross as in the case described should occur with normal electrocardiographic findings over several years. The reason for the lack of cardiographic changes in this patient is obscure. This case emphasizes that the relationship between the electrocardiographic features and pathological changes in patients with *angina pectoris* is still not fully worked out and presents an intriguing problem. It is somewhat strange that some relief was not obtained by left cervico-dorsal sympathetic blockade; although pain may still occur after unilateral sympathetic denervation of the heart, it is stated to stop at the mid-line (White *et alii*, 1952).

Thirdly, the danger of prolonged anæsthesia in patients with coronary ischæmia is stressed. Surgical operation would not have been advised if we had been sure of the correct diagnosis.

Summary.

The clinical history is given of a man who complained of chest pain, intermittent claudication and muscular ischæmic pain in his left arm for ten years. The only definite abnormal sign was the presence of poor pulses in the left arm. Repeated scalar electrocardiography failed to show any evidence of coronary heart disease.

Death followed surgical exploration of his thoracic outlet region. Necropsy revealed gross coronary atheroma and occlusion of the origin of the left subclavian artery, also due to atheroma.

This history is recounted to stress (*a*) the diverse manifestations of atheroma, (*b*) the occasional unreliability of electrocardiography in the diagnosis of *angina pectoris* even with gross coronary atheroma, and (*c*) the hazards of anæsthesia and surgical operation in patients with severe coronary heart disease.

Acknowledgements.

Our thanks are due to Dr. M. D. Silberberg for supplying his case notes and helpful suggestions on the preparation of this paper, to Dr. Eric Clarke for referring the patient for investigation, and to Dr. T. E. Lowe for advice and criticism. The necropsy was performed by Dr. K. M. Bowden (Government pathologist), and the histological examination by Dr. A. V. Jackson (pathologist, Alfred Hospital, Melbourne).

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TWO CASES OF PRIMARY AMENORRHŒA.

By J. H. STUBBE,
Perth.

THE following case reports are thought worthy of publication, illustrating as they do entirely different types of developmental anomalies. Remarkably, the two patients presented in the same week; indeed, they occupied adjacent beds in hospital.

Case I.

Mrs. A., aged thirty-four years, a barmaid, complained of right-sided lower abdominal pain. She stated that she had never menstruated, and had always been lean and without breasts. She had been twice married, and claimed to have had mutually satisfactory relations with both husbands. One older sister had three children. The patient was of medium height, and slim, and her physique being of completely android type. Her chest measurement at the nipple line was 29 inches. However, her voice and hair distribution were of female type. To my gratification, deep tenderness was present over McBurney's point. The vulva was infantile, but the clitoris was rather large; the vagina was roomy, but the cervix was tiny. No uterine body or adnexæ could be palpated.

On June 29, 1954, at laparotomy, the pelvic organs were seen to be completely rudimentary. The uterus was merely a muscular thickening in the centre of the broad ligament. The ovaries were normally sited, but were no larger than peas; no *corpora lutea* or *corpora albicantes* were present. Although all the classical criteria of Turner's syndrome were not present, this was thought to be a case of primary ovarian agenesis.

Since operation, dienœstrol has been exhibited in a dosage of nine milligrammes twice daily. Her mammæ are rapidly developing; on September 10 her bust measurement at the nipple line was 34·5 inches. On September 21 she reported a slight hæmorrhagic discharge *per vaginam*.

Comment.

Should such replacement therapy be permanently maintained, as in diabetes or myxœdema? Will her genital organs attain normal stature and function? Is there any real fear of a carcinogenic effect in such therapy?

Case II.

Miss B., aged thirty years, a senior floor-girl in a large department store, was referred by Dr. F. Ebell. She said

that she had never menstruated and had no vagina. She was contemplating marriage and desired to be capable therein; she had female libido, but had never attempted intercourse. One sister had two children. She was of pleasing gynæcoid habitus. A tumour was palpable in the left loin above the iliac crest. The vulva and clitoris

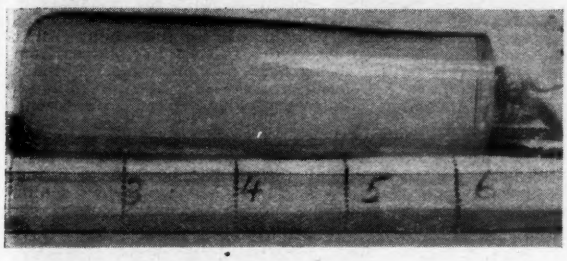

FIGURE I.

appeared normal, but the vagina was represented by a dimple, perhaps a quarter of an inch deep. On rectal examination a small nodule was palpated in the position which a cervix should occupy.

On July 5, 1954, laparotomy was performed. The tumour in the left loin was a dermoid cyst, the size of a billiard

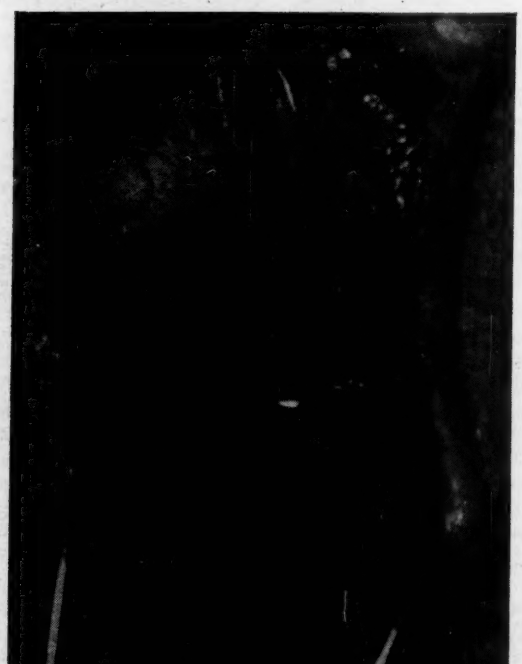

FIGURE II.

ball, arising from an undescended left ovary. The right ovary was similarly sited, but was not cystic; both ovaries were large and elongated, and contained *corpora lutea* and *corpora albicantes*. The uterus was rudimentary, and of a *bicornis unicollis* type. The condition was apparently due to failure of the gubernaculum, and of proper development at the Müllerian duct. Ovulation was established, but the absence of normal endometrium fortunately prohibited menstruation and hæmatometra.

With the assistance of Mr. G. D. Henderson, who manufactured a "Perspex" obturator (Figure I), of Mr. G. S. Pestell, who prepared the Thiersch graft, of Dr. V. T. White, and of Dr. O. C. Hood and Dr. N. A. Gunn, who administered the anæsthetic, an artificial vagina has been created (Figure II). It is completely epithelialized, and easily admits the complete large blade of an adult Sims speculum. The patient has resumed her usual occupation and wears her obturator only at night. She is pleased with the cosmetic result, and has no doubts of future marital competence.

Reviews.

Study Guide for Clinical Nursing: A Co-ordinated Survey Integrated with Essentials of the Basic Sciences. Prepared under the direction of Emily C. Cardew, R.N., M.S.; 1953. Philadelphia, London and Montreal: J. B. Lippincott Company. 9″ × 6″, pp. 576. Price: 64s. 6d.

THIS book is set out in such an unusual style that a description of its make-up is necessary. Firstly, it is divided into the following four sections: (i) Nursing Care of Adult Patients with Medical and Surgical Disorders. (ii) Nursing Care of Children. (iii) Nursing Care of the Psychiatric Patient. (iv) Nursing Care of Mothers and the Newborn.

Each of these is divided into smaller chapters which contain a list of study suggestions, and these are followed by a series of questions termed "Background Review Questions", and also a series of "Questions for Discussion and Student Projects"; finally there is a "Patient Study" with more questions relating to the patient's case. In a pocket attached to the back cover is a booklet of answers.

In the patient study, the style is to tell the story of a case history, one of which concerns the problem of "The Man Who Couldn't Breathe". The questions which follow have a choice of answers set out and the student places a cross in the square opposite the one that is considered to be correct.

From the foregoing it is hoped that some idea is conveyed of the rather unique style of the work. Each section is written by a professor or director of nursing from various well-known United States universities; and while the work is very American in colour, yet it can be used to advantage anywhere.

The preface states that the book is written for student nurses, graduate nurses and clinical instructors, and that it has a "patient-centered approach". As it consists of 563 pages, it can readily be seen that the work is of considerable size, and containing as it does a discerning survey of important topics, it should prove popular with all nurses and their instructors.

Emergencies in Medical Practice. Edited by C. Allan Birch, M.D., F.R.C.P.; Fourth Edition; 1954. Edinburgh and London: E. and S. Livingstone, Limited. 8½″ × 6″, pp. 622, with 143 illustrations, nine in colour. Price: 32s. 6d.

SOME idea of the completeness of this volume can be gauged from the fact that even though it is published by the House of Livingstone in Great Britain, yet it contains the addresses and telephone numbers of all the laboratories in Australia, State by State, where tiger snake antivenines can be obtained. Although Dr. Birch edits the book there are in all 23 contributors, and as each is a specialist in his own field, each topic is dealt with authoritatively. In the preface, medical emergencies are defined as all those except ones needing surgical intervention, and the critical reader will be unable to find any emergency that is omitted. Thus there are chapters devoted to emergencies in the tropics, emergencies on board ship or in an aeroplane as well as the everyday happenings nearer home, whether they are bites, poisonings, burns or frost-bites. An ingenious method for removing a tight wedding ring with the aid only of a piece of string is described, and so, too, is the efficient Holger Neilson method of resuscitation which has largely replaced the old Schäfer method.

All doctors can learn from the chapter headed "Hazards of Medical Procedures" which deals with all topics such as broken needles, air embolism and reactions to local and

general anæsthetics. Chapters are also devoted to the medico-legal emergencies, to those associated with childhood and infectious fevers, and to those concerned with each special bodily system, to mention only some.

This is indeed a work which all doctors, no matter in what field they work, should have by them, and the appearance of four editions in six years proves its worth.

Living Happily with a "Heart". By Tom Clarke; 1954. London: Victor Gollancz, Limited. 7½" × 4½", pp. 132, with one illustration. Price: 7s. 6d.

THE author was a prominent busy London journalist and broadcaster when smitten with what is often termed the doctors' disease—coronary thrombosis.

In this book he recounts his story of the first and then a subsequent attack, and goes on to tell how he readjusted his life to meet the restrictions which his heart placed upon his activities. He went from the busy city to live in the country, and he extols the virtues of relaxed living which have given him a decade of happy years to look back on since the day he developed "a heart".

The reader might think this is just another story about illness—but it is in fact far from that and is written with a charm akin to that of Lamb's delightful "Essays of Elia". Although it is perhaps the smallest of Tom Clarke's well-known books, being so intensely personal, it has a greater appeal than some of his major efforts.

This is a book that all thoughtful readers will enjoy and one that will also hearten those who have suffered from a coronary thrombosis.

Primer of Allergy: A Guidebook for Those Who Must Find Their Way Through the Mazes of This Strange and Tantalizing State. By Warren T. Vaughan, M.S., M.D., revised by J. Harvey Black, M.D.; Fourth Edition; 1954. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. 8" × 5½", pp. 192, with 23 illustrations. Price: 44s. 9d.

THIS book is written to be read by patients to give them as full as possible an insight into their own allergy. It is difficult to make such a complex subject simple enough for laymen, and in parts of the book the language is perhaps too medical.

The causes of allergic reactions are all detailed and much good advice is given in regard to avoidance of sensitizing agents. Moreover, the patient is warned not to be discouraged if not cured overnight and exhorted not to rush off and try a series of "cures" that well-meaning friends may advise or which may be advertised in the popular Press. A very useful diary page is set out which lists many foods and also a suggested form for listing the day's activities and contacts is printed, both of which will aid the physicians to track down the offending allergen. A copy of this book in the surgery could be handed to patients for guidance, for it will give them first-hand information on the part they can play to help their doctor rid them of their disability.

Fundamentals of Anesthesia: Prepared under the Editorial Direction of the Consultant Committee for Revision of Fundamentals of Anesthesia, a Publication of the Council on Pharmacy and Chemistry of the American Medical Association. Third Edition; 1954. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 9½" × 6½", pp. 296, with 81 illustrations. Price: 57s.

THIS third edition has been prepared under the editorial direction of the Consultant Committee for Revision of Fundamentals of Anæsthesia. It is published for the Council on Pharmacy and Chemistry of the American Medical Association. The earlier editions were published by a Sub-committee on Anæsthesia during the war for the express purpose of providing instruction for medical officers and others in the details of anæsthesia and the care of patients with circulatory and respiratory depression.

The new book is larger than the old, the latter having been largely rewritten and chapters having been added on certain new developments. The list of contributing authors contains the names of many anæsthetists known the world over for their prowess in their special fields. The book is well illustrated, very well printed and set out like the earlier editions under numerous headings and numbered sub-headings. It is a collection of facts which are presented without frill or padding.

One of the new sections on physics and chemistry follows the form suggested and much of the substance in the book published by McIntosh and Mushin. Very great detail is given for certain procedures which we would consider elementary; for instance, most minute detail is given about the use of ordinary open [illegible]ach step in the technique being described fully. Th[illegible]t detail also appears in connexion with many other t[illegible]ques.

In view of the foregoing it [illegible]disappointing to note that such important and everyday techniques as the control of respiration and the use of relaxants are mentioned without sufficient detail to afford any instruction in their use. The combination of controlled respiration with the relaxants in conjunction with the lightest forms of anæsthesia has become so much an everyday matter in this country that the omission of detailed description of these techniques from a book of this nature seems hard to understand. Perhaps the authors have not decided so far that these techniques are within the range of the book, which claims to deal with fundamentals.

With these notable exceptions, "Fundamentals of Anæsthesia" provides an excellent detailed setting-out of the basic knowledge and technique of anæsthesia and is what it was designed to be, a valuable asset to those learning or teaching anæsthesia.

Histopathology of the Skin. By Walter F. Lever, M.D.; Second Edition; 1954. Philadelphia: J. B. Lippincott Company. Sydney: Angus and Robertson, Limited. 9" × 6½", pp. 536, with 281 illustrations, eight in colour. Price: £6 9s.

ONCE again in the second edition the author is to be congratulated on the excellence of his text and quality of his photographic reproductions. The 281 illustrations are uniformly good. The contrast between black and white and coloured plates is great and much in favour of the latter. Only when processing and reproduction of coloured photographs becomes less expensive can one hope to see the great majority of histological reproductions in colour.

The differential diagnosis at the end of each description is invaluable. It is of great use for teachers and students. The text throughout is well prepared, well produced and more readable than are most pathological descriptions; it is well supported by an extensive bibliography arranged at the end of each chapter.

We find little to criticize in the minutiæ of Dr. Lever's histological descriptions which are as accurate and understandable as possible. His classification of such diseases as the lipoidoses is acceptable to dermatologists and physicians in this country. Verbiage is at a minimum. This book is an essential component of a dermatologist's library, and the readiness with which details can be obtained render it a most satisfactory reference book.

Diseases of the Skin. By Oliver S. Ormsby, M.D., and Hamilton Montgomery, M.D., M.S.; Eighth Edition; 1954. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 10" × 6½", pp. 1504, with 768 illustrations, 18 in colour. Price: £11 16s. 6d.

A NEW EDITION of "Diseases of the Skin", by Ormsby and Montgomery, is always welcomed by dermatologists. It has become the reference book of choice in the English language. It is to be regretted that volumes of a comparable type have not been kept up to date by British dermatologists. The original author, Dr. Ormsby, died whilst the proofs of this eighth edition were being read.

The writers have been fortunate in their choice of four eminent contributors: Edward D. DeLamater, M.D., Ph.D., Eugene M. Farber, M.D., M.S., Robert R. Kierland, M.D., M.S., Walter C. Lobitz, junior, M.D., M.S. The last edition was published in 1948 and the present one follows the same type of set-up; it includes many of the old illustrations, but its value is enhanced by the inclusion of 117 new ones.

All the old chapters have been modernized and some have been completely rewritten. Space does not allow for any detail, but attention should be especially directed to the following: "The Chemistry and Physiology of the Skin" (Lobitz), "Cutaneous Vascular Disorders" (Farber), "Serology and Treatment of Syphilis" (Kierland), "Diseases due to Plant Parasites" (DeLamater). In addition to this revision several new or rare diseases are now included. The main omission is that there is no guide to the dosage with X-ray therapy. This form of therapy is the main weapon in the armamentarium of the qualified dermatologist. Surely it is not asking too much for such an expensive volume to include details from such experienced authorities. No text-book, however comprehensive, can be completely up to date, and one can find no reference to "Atebrin" or "Chloroquin" in light sensitivity. This volume is a "must" for a skin specialist who does not possess an "Ormsby". The owner of a 1948 edition will have to consider if he is prepared to pay nearly twelve pounds for this excellent publication so full of recent information and references. If money is no object, he should not hesitate.

The Medical Journal of Australia

SATURDAY, FEBRUARY 26, 1955.

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TUBERCULOSIS IN IRELAND.

THE Medical Research Council of Ireland has recently issued a report of the National Tuberculosis Survey which was carried out during the period 1950 to 1953. The report is by Dr. James Deeny, chief medical adviser of the Department of Health. All students of preventive medicine, and of tuberculosis in particular, will be interested in this report, because tuberculosis has commonly been thought to be more prevalent in Ireland than in most other countries. As a matter of fact, John B. McDougall, in his book on tuberculosis which he calls "A Global Study in Social Pathology" (1949), refers to F. C. S. Bradbury's "Tyneside Inquiry" and quotes him as stating that the Irish had a racial susceptibility to tuberculosis. It is doubtful whether most students of the subject would agree with this view, and the present report shows without doubt that the problem of tuberculosis in Ireland is becoming less acute. Indeed, the author of the report is sufficiently optimistic to look forward to the day when tuberculosis will have disappeared from Ireland. The National Survey of Tuberculosis was undertaken as part of the intensified campaign against the disease which has been in progress for the last ten years. This campaign has included short-term and long-term programmes for the provision of new bed accommodation, the development of B.C.G. vaccination, the organization of major chest surgery facilities, the establishment of a mass radiography service and the provision of allowances for patients. These features of the campaign have been supplementary to the elaboration and extension of the facilities for diagnosis and treatment and epidemiological control already provided by local and voluntary authorities. It was in May, 1950, that the Department of Health, by direction of the Minister, Dr. M. C. Browne, approached the Medical Research Council of Ireland with a proposal that it should sponsor a tuberculosis survey. The Department offered to defray the entire cost. The Council accepted the proposal and the chief medical adviser to the Department of Health was seconded for the Council's service to take charge and direct the survey. It was decided to commence the survey with reference to the history and folklore of tuberculosis in Ireland and then to proceed with the study of deaths. A recent period was selected which would be long enough to show general trends, would provide sufficient numbers for worthwhile analysis, and would enable the wartime increase in deaths to be examined. The period chosen was the twelve years from 1938 to 1949 inclusive.

The section on tuberculosis in Irish folklore, though full of interest, cannot be more than mentioned in this summary. Suffice it to observe that affected families have for generations been thought to be "delicate" or "not to be strong". Until quite recently there has been insufficient appreciation of the part played by infection in the spread of the disease or of any other factors in its causation. The history of tuberculosis in Ireland must likewise be but briefly referred to. For more than 100 years the country has experienced a national epidemic of the disease. This commenced in the early nineteenth century, reached its peak at the beginning of the twentieth, and is now on the wane and nearing its end. The gradual disappearance of the disease in the first forty years of this century has been largely due to increasingly favourable ecological factors, but influences such as increased urbanization, which favour the spread of infection, have tended to offset the full effects of more favourable circumstances of living. Within the last ten years considerable changes have taken place in the pattern of tuberculosis in Ireland. It has become less deadly, and violent fulminating forms are rare; this may be due to the increasing immunity of the population or to the decrease in virulence of the disease. At the present time, increased sanatorium accommodation offers improved facilities for the isolation of infectious persons, radiography offers a means of early diagnosis, B.C.G. vaccination can offer a method of enhancing resistance to the condition and antibiotic drugs offer a means of cure. The disease has now to be viewed in the light of these changes, and methods have to be altered, not only to deal with its changing features, but to ensure that new techniques are employed to the greatest advantage. The changes taking place in tuberculosis in Ireland are occurring in other countries as well. In other countries there has been a change in the age of onset from adolescence to middle-age, but this has not yet occurred in Ireland, and deaths from the disease are largely concentrated in the young adult age group, particularly among young women, though a secondary emphasis is noted amongst older men. Urban influences, which provide increased opportunities for infection and favour stress, are the chief factors affecting the distribution and occurrence of deaths.

From the tuberculosis registers throughout the country a total of 11,642 patients were classified as "cases" of pulmonary tuberculosis. There were 6034 males and 5608 females. Data on the cases were collected and recorded on a standardized chart. More than 85% of the patients were interviewed and a study was made of each patient. It is

interesting to note that of 9276 patients whose chests were examined by X rays and of whom skiagrams were available, no less than 3485 had cavities in the lungs. There were 2788 non-pulmonary cases of tuberculosis. The patients with pulmonary lesions had 38,151 family contacts. These numbers are regarded as small in relation to the total population of the country, but this fact is offset by the severity of the disease. One of the most significant facts noted was that in many instances the mechanical damage, which had already taken place in the lungs before a diagnosis was made, was sufficient to impair function to a very large extent and to affect permanently the lives of patients and their ability to work. With the provisfon of increased bed accommodation and the introduction of antibiotic drugs and other forms of treatment, the usual tendency in many infections in Ireland for death to occur rapidly has been reversed; more patients are surviving and the death rate is falling. Unfortunately, this trend is not yet being accompanied by a sufficient fall in the number of new cases or by an increase in the number of effective cures. Most of the sputum-positive patients are safely in sanatoria under treatment and are unable to spread the infection. Those who are at home under domiciliary care are insufficient to account for the new infections occurring and for the present spread of the disease. The present spread is stated to be due to the number of unknown infections, either of the chronic, long-term undiagnosed form, or in persons who are in the early stages of the disease before symptoms bring them to their medical attendants. The typing of the strains of tubercle bacilli was carried out in 903 instances. Of 860 strains from pulmonary sources, nine, or 1%, and of 43 strains from non-pulmonary infections, five were of the bovine type. In regard to bacteriological studies, we read that it is important to establish standards of normal characteristics, since the advent of antibiotic drugs would seem likely to affect the nature of the organism. A few of the difficulties to be met with in the future in regard to bacteriological control were to some extent appreciated. Further, the necessity for a great increase in the amount of bacteriological work on the infectivity of the patient and for sensitivity tests of the organisms to the antibiotic drugs used was also made clear. Mass X-ray community surveys were carried out in six centres (a large town, a small town and four typical rural parishes), and in five months 21,500 people were surveyed. In the six areas, an average of 67·5% of the population was examined. In all areas, 88·5% of females under the age of forty-five years were examined and 82·5% of all males. The greatest difficulty was experienced in securing the cooperation of persons over forty-five years of age. In summary, the results of the National Tuberculosis Survey are described in the following terms: "The incidence of tuberculosis is much more limited in comparison with the size of the population than was previously believed. The nature of the disease is such, however, that the majority of the cases under present circumstances are already serious on first recognition of the diagnosis. The disease is limited very largely to occurrence in young adults living in cities and towns. It is particularly likely to occur in families with previous deaths or cases, and it is much more prevalent in some industries than in others."

The recommendations of the report are set out under three headings: general, case-finding and domiciliary care. In regard to coordination, it is stated that the time is ripe for coordination and agreement in effort of the various services operating against tuberculosis. These include those provided by practitioners and consultants and by the voluntary and local hospitals, by the specialist tuberculosis services and institutions provided by county boroughs and county councils, and by specialist regional services and regional institutions dealing wholly or in part with tuberculosis. Emphasis is laid on the need for a national recording system. The fact that different recording systems were used in various county and city health services created one of the major difficulties of the survey. If a uniform national recording system was introduced, information would be capable of statistical analysis by machine methods, and it is stated that the confidential nature of the records should be fully preserved. Case finding is discussed under the headings of "In the Home", "Employment" and "In the Community". For each patient, "defined as a case", there are on the average between three and four contacts in each family. The amount of attention required varies at different ages, but roughly one-third of the number of contacts, that is, people in the older age groups, require only a radiological investigation to ensure that they are not suffering from the disease, and this ordinarily should suffice. For another third of the total, ranging from infants to those about to leave school, B.C.G. vaccination, a yearly examination and facilities for occasional preventorium care are required. For the middle group of adolescents and young adults, the care necessary depends on circumstances. At least one-third of all new infections may be expected to originate from amongst 38,151 known contacts in the comparatively small number of families. "There is no other branch of tuberculosis work in this country which is likely to yield such a result as the intensive care of family contacts of cases, particularly of those contacts in the young adult ages." The need for the protection of persons engaged in industry is realized. For their protection, it is recommended first of all that the tuberculin test, and if necessary B.C.G. vaccination, of all young persons should be carried out before they commence employment. Secondly, an annual X-ray examination of all persons, particularly young adults engaged in industry, is recommended. Thirdly, it is suggested that X-ray examination of insured persons should be undertaken when they are called for medical referee examination under the National Health Insurance Scheme. In rural areas, B.C.G. vaccination should continue at an increased rate while the urban surveys are being completed; and it is thought that rural areas should then be surveyed by mass radiography according to parishes. Recommendations in regard to domiciliary care of patients are discussed under two headings, first of all "on initial diagnosis" and then "the continuing case". It is not intended to discuss it in much detail, but we would point out that the general practitioner is stated to be so important in case-finding and in relation to the health of the family of the patient that he must be retained in the team and his interest and assistance should be preserved. The main obstacles to adequate care and supervision of the patient at home are usually the discouragement and

apathy of the patient himself, as a result of which many cease to attend or to accept advice. Two important features specially mentioned in connexion with the domiciliary case are the psychological features present in the continuing case and the need for increased appreciation of the danger of the domiciliary patient acting as a disseminator of infection.

It will be realized that only the outline of this report has been described. What has been presented, however, should stimulate the interest of all who have to do with pulmonary tuberculosis, and especially of the general practitioner whose place in the prevention and treatment of the disease is regarded as of tremendous importance. Every reader will be able for himself to compare features described in this report from Ireland with conditions existing in Australia.

Current Comment.

REGIONAL ILEITIS.

In 1932, B. B. Crohn, L. Ginzburg and G. D. Oppenheimer[1] described a disease for which they proposed the name "regional ileitis". This name has been retained by most writers on the subject. The name "Crohn's disease" has also been used a good deal, although we have not been able to find who introduced it. With commendable modesty, Crohn, in a recent article with H. D. Janowitz as co-author, ignores the eponym, retaining the term "regional ileitis".[2] In their original description, Crohn, Ginzburg and Oppenheimer stated that the disease was one of the terminal part of the ileum, affecting mainly young adults and characterized by a subacute or chronic necrotizing and cicatrizing inflammation. "The ulceration of the mucosa is accompanied by disproportionate connective tissue reaction of the remaining walls of the involved intestine, a process which frequently leads to stenosis of the lumen of the intestine, associated with the formation of multiple fistulas." The clinical features of the disease were described as resembling those of ulcerative colitis—namely, fever, diarrhœa and emaciation, leading eventually to obstruction of the small intestine. The constant occurrence of a mass in the right iliac fossa usually required surgical intervention for its resection. The terminal part of the ileum was alone involved. The process began abruptly at, and involved the ileo-caecal valve in its maximal intensity, tapering off gradually as it ascended the ileum for from eight to twelve inches. The familiar fistulas led usually to segments of the colon forming small tracts communicating with the lumen of the large intestine. Occasionally the abdominal wall anteriorly was the site of one or more of these fistulous tracts. The ætiology of the process was unknown. Its course was relatively benign, all patients who had survived operation being alive and well. This original description was based on the study of 14 cases of which Crohn, Ginzburg and Oppenheimer had first hand knowledge, but it was apparent from the discussion which followed the presentation of their paper before the section on gastroenterology and proctology of the American Medical Association that other surgeons had observed the condition.

Since 1932 a great many papers on regional ileitis have appeared. Some aspects of the disease have been elucidated, but a good deal still remains obscure. Crohn and Janowitz's recent paper gives an interesting review of the present position. Their original small series of 14 cases of ileitis involving only the terminal segment of the small bowel has now grown to 562 cases of non-specific granulomatous enteritis involving the distal part of the ileum, the ileum and jejunum, the duodenum and probably, although rarely, the stomach. Crohn and Janowitz ask themselves whether regional ileitis, as originally described, was an accurate picture of a new clinical and pathological entity. They are satisfied that as a clinical picture it was, pointing out that even today the original notations in relation to symptoms, complications, fistulas and course have been but little amended. For the condition as a pathological entity, singular and discrete, they consider the evidence perhaps less convincing; they state that although the peculiar lymphatic blockage, intense follicular regeneration and miliary-like tubercles with giant cell system formation are strictly characteristic of enteritis involving the terminal part of the ileum, the similar pathological characteristics of non-specific inflammatory involvement of other segments of the small and, rarely, the large intestine deny to ileitis existence as a pure and isolated pathological entity. On the question of terminology, they consider that, strictly speaking, regional enteritis is the most acceptable generic name, but regional ileitis has an historical appeal and covers the pathological features of the vast majority of the cases.

The ætiology of the disease remains unknown, although certain related facts have emerged. Crohn and Janowitz have, in their own material, encountered striking family constellations of this illness at least eleven times, but they state that there does not exist sufficient material for any attempt at genetic interpretation. No support has been forthcoming for a bacterial origin of the disease and there are also great difficulties in ascribing it to a virus. A non-caseating, tubercle-like granuloma is a characteristic, if not pathognomonic feature of the disease; but tuberculosis seems to be clearly ruled out, and sarcoidosis also is considered unlikely to be the cause. An allergic basis has been suggested, but the point has not been proved. A granulomatous reaction to severe non-penetrating trauma to the abdomen has been reported in certain cases, but appears to be exceptional. A psychosomatic cause has also been proposed, but Crohn and Janowitz's impression is that, unlike the situation in ulcerative colitis, superficially at least emotional stress does not appear to be a major factor in precipitating ileitis. This attitude was broadly supported in a review by R. W. Crockett of 32 cases of regional ileitis at the General Infirmary at Leeds.[1] He found that routine psychiatric examination gave no substantial support for the suggestion that emotional stress was a major ætiological factor in Crohn's disease. However, he stated that the examination did confirm that in some patients emotional stress played a material part in the formation of symptoms, and in that sense might be accepted as a factor capable of affecting the course of the illness, although ordinarily within physiological expectations. Crohn and Janowitz go on to point out that lymphangiectasis or granulomatous lymphatic involvement appears to be a central pathological feature of the disease. Animal experiments indicate that materials present in the intestinal stream may enter the ileal lymphatics and set up productive inflammatory reactions which resemble those of ileitis. It has been suggested that some local disturbance in fat metabolism or abnormality in fat absorption is involved in the characteristic tissue reaction of ileitis. Crohn and Janowitz suggest, just as a speculation, that a noxious agent is present in the intestinal stream, being either concentrated or absorbed by the lymphatics that are so rich at the region of the ileo-caecal valve.

Turning to the pathological aspects of the disease, they point out that it was originally described as a pathological entity. Subsequently, the non-specificity of the lesion has been stressed in some quarters, but Crohn and Janowitz are able to quote the work of various investigators which confirms the histological identity of regional ileitis and its clear morphological differentiation from the pathological process of ulcerative colitis. It is agreed that because some combined forms of small intestinal and colonic

[1] *J.A.M.A.*, October 15, 1932.
[2] *J.A.M.A.*, November 27, 1954.

[1] *Lancet*, May 10, 1952.

inflammatory disease do coexist, the fundamental significance of the pathological separateness of the two entities is not clear.

Little comment is necessary on the clinical aspects of the disease, but mention may be made of the apparent increase in incidence and of the tendency in recent years for abdominal wall fistulas to disappear. Crohn and Janowitz also point out that the great incidence and diagnostic significance of perianal or perirectal abscesses and fistulas were not apparent in their original communication, but have since become so to all observers. They state that since these arise from infections in the rectal crypts of Morgagni and almost never from the small intestine directly, this would seem to indicate that the noxious agent is in the fæcal current, accumulating behind the ileo-caecal valve or the anal sphincter. The one major addition to our knowledge of the clinical features of the disease since the original description is stated to be the recognition that this granulomatous process not only occurs in the terminal part of the ileum but can occur in the jejunum alone or in combination with the ileum. Recent evidence has also been accumulating of lesions in the duodenum, and on rare occasions in the stomach. This widening of the distribution of the basic disease has accordingly widened the clinical manifestations. Nevertheless, in the vast majority of patients suffering from regional enteritis the disease is localized in the terminal part of the ileum, and most recurrences after either shortcircuiting or resection operations occur in the new terminal part of the ileum. On the other hand, granulomatous involvement of the colon is rare.

Crohn and Janowitz go on to point out that there are large gaps in our knowledge of the life history of regional ileitis, especially of the untreated disease. This naturally makes it more difficult to give a prognosis with certainty and to evaluate a treatment. It is established, however, that spontaneous remissions occur in some cases, although few patients go on to complete healing spontaneously. There is evidence to show a tendency towards spontaneous healing, but the factors that influence this reparative process are not understood. Diagnosis, apart from exploratory laparotomy, depends on radiographic findings. There is no specific therapy for the disease; all currently employed methods including the use of cortisone and ACTH are simply supportive. Crohn and Janowitz state that the high rate of recurrence of ileitis after the shortcircuiting or the resection type of operation is leading to a reevaluation of the criteria for operation. The current trend of thinking is to defer operation until the pathological process appears to be quiescent and to restrict surgery to the complications of the disease, namely, obstruction and formation of fistula.

HYPERTENSION.

THE condition known as hypertension has been discussed in these columns on many occasions and abstracts from medical literature on the subject have appeared in the abstract section from time to time. It might appear that any further reference to the subject would be unnecessary, but when we find that so august a body as the Association of Physicians of Great Britain and Ireland chose a discussion on hypertension as one of the subjects for its annual meeting at Dublin in 1954, no further excuse need be offered for another reference to it. Abnormal readings of blood pressure are extremely common, though in truth it is not particularly easy to define what is normal and what is abnormal. When we come across a patient with a particularly high systolic reading we naturally keep him under observation until we discover whether the high reading is, as it were, a flash in the pan or whether it is indicative of that sinister condition known as essential hypertension. Some of the many methods of treatment of essential hypertension were set out recently in the issue of December 4, 1954, in the light of the discussion that took place at one of the staff meetings of the Mayo Clinic.

[1] *Quart. J. Med.*, October, 1954.

At the Dublin meeting of the Association of Physicians of Great Britain and Ireland the discussion was opened by Professor G. W. Pickering,[1] who dealt with basic considerations affecting the management of patients with hypertension. He reminded his audience that, broadly speaking, patients suffering from this condition fell into two classes. The first comprised those in whom a specific cause for the hypertension could be recognized and removed, and the second those in whom this could not be done. The first group of conditions included coarctation of the aorta, phæochromocytoma, unilateral renal disease and probably Cushing's syndrome. Professor Pickering discussed hypertension in the first group and he showed that in human hypertension, as in experimental hypertension, removal of the original cause of a prolonged hypertension might not reduce it substantially. He said, for example, that removal of a unilaterally diseased kidney, which was well recognized as an effective method of reducing high blood pressure, would work only if the other kidney was free from gross disease, usually pyelonephritis. He had had experience of ten cases of apparent unilateral pyelonephritis in which the arterial blood pressure had been substantially reduced in only five. Particular interest is attached to Professor Pickering's second group in which the cause could not be removed. This included nephritis, bilateral chronic pyelonephritis, polycystic kidney and *polyarteritis nodosa*. He said, however, that the great majority of cases were classified by exclusion as essential hypertension and that they constituted the chief problem in the management of the condition. He stated two conclusions about the nature and ætiology of the condition. The first was that so-called essential hypertension was not a well-defined condition, but merely represented the higher ranges of arterial pressure for which no discrete cause could be found. The second was that its cause was to be found in the genetic and environmental factors which affect the population as a whole. Two factors, heredity and age, were partly understood, but the effect of environment was not understood at all. He explained that the evidence for these statements was derived from measurements of the casual arterial pressure in a sample of the population and in first degree relatives of subjects with so-called essential hypertension, and subjects with normal pressures respectively.

Discussing how far hypertension caused disability and death, Professor Pickering said that records of insurance companies showed that expectation of life was inversely proportional to pressure and that this relation held down to quite low values. It had been shown by Volhard and Fahr that the whole course of hypertension might be stable for many years or might be rapidly progressive to renal failure within the year. The clinical distinction between the two courses was based on the appearance of the ocular fundus. Extensive retinal changes were found in the malignant form, but in the benign form either there was no retinal lesion apart from vascular change, or the retinopathy tended to be unilateral and was without disk swelling and with small and sharply defined exudates. Referring to the arteriolar necroses found in the kidneys and other organs, Professor Pickering explained that these necroses stopped the blood flow and killed the tissues supplied by the arterioles. There was an increasing body of evidence suggesting that an important factor in the production of the lesions was the intensity of the hypertension, so that the malignant phase was essentially a consequence of the degree of hypertension rather than of its kind. In the malignant phase, considerable reduction of arterial pressure was the urgent and first aim of management, because this often seemed to arrest the course of the arteriolar lesions which would otherwise kill the patient. In the benign form indications were less clear. Professor Pickering chose a happy phrase when he said that all treatments that were effective in reducing arterial pressure had a "nuisance value" which might convert the patient into a neurotic invalid. It was the essence of good management that a balance should be struck between the gains and the losses that a particular therapy was likely to confer.

Professor J. McMichael discussed the place of hypotensive drugs. He regarded parenterally administered

methonium compounds as the most effective agents. Phasic reduction of blood pressure produced amelioration in retinitis, in cardiac failure and in headache. Occlusive vascular changes were not affected. He had obtained favourable results in the malignant hypertension of 15 out of 22 patients; these 15 had survived beyond nine months and six at the time he spoke were living after more than two years. Notable regression of retinitis had taken place in all instances, and in the patients surviving longest it had disappeared completely. He had found that other hypotensive agents such as veratrum, hydralazine and ergot alkaloids were less satisfactory. He thought that rauwolfia alkaloids might find a place in the treatment of less severe hypertensive illness. Statistically satisfactory evidence of drug efficacy in benign hypertension, however, was lacking.

It is interesting to note that F. A. Finnerty, junior, has recently made a study of 89 ambulatory patients suffering from hypertension.[1] He investigated the value of treatment with *Rauwolfia serpentina*. This is a long and detailed study which unfortunately cannot be described in full. Two of his conclusions, however, may be quoted since they bear on what was said by Professor Pickering. Finnerty found that rauwolfia is of value in mild to moderate degrees of hypertension and that results obtained with the drug are superior to those obtained with mild sedation alone. In the more advanced cases, the drug should be supplemented with more potent hypotensive drugs such as veriloid, hexamethonium, or other ganglionic blocking agents and/or apresoline. Finnerty found no appreciable benefit in congestive failure and no valid evidence that the status of the hypertensive cardiovascular disease was changed appreciably by rauwolfia alone. However, on combination therapy, congestive failure was found to clear in several instances, and he stated that there was reason to believe that simple lowering of the blood pressure slowed the progression of vascular disease and might in time allow regression of organic changes.

Observations may also be quoted which were made by R. W. Wilkins in a seminar on anti-hypertensive drugs.[2] Wilkins concluded that rauwolfia was a distinct addition to drug therapy of hypertension. While it was only moderately hypotensive, it filled a previously unfilled need, namely, for a mild, slowly and progressively active agent with few or no side effects. It also met much better than the ordinary sedative the need for a sedative-like drug. He said that perhaps the greatest usefulness of rauwolfia was in combination with the more powerful hypotensive drugs with which it served as an adjunct or background agent, smoothing the course and increasing the hypotensive effects of the more powerful agents.

Returning to the Dublin discussion, we note that Professor Robert Platt discussed the optic fundus as a guide to management. He described the following five kinds of change: (i) Vascular changes such as constriction, tortuosity and venous compression. (ii) Vascular accidents, such as arterial and venous thrombosis, central or peripheral. (iii) Hypertensive neuroretinopathy which was the most important evidence of the malignant phase in hypertension and demanded the use of measures to reduce blood pressure unless such treatment was otherwise contraindicated. (iv) Arteriosclerotic retinopathy, such as hæmorrhage and hard exudates without papilloedema, which overlapped with the previous group, but if typical, denoted a more gradual and not necessarily malignant course. (v) Papilloedema, which might occur without the development of diffuse neuroretinopathy in some instances.

Passing over references made by different speakers to the dietary management of hypertension, to its surgical treatment, and to the effects of resection of coarctation of the aorta on the blood pressure, we must conclude by referring to remarks made by Dr. Maurice Sokolow on the psychological aspects of the treatment of hypertension. Dr. Sokolow said that although physicians had for years appreciated that hypertensive patients frequently showed maladjustment of their personality, the role of psychological factors in hypertension had not been established either in terms of ætiology or in regard to their effect on the course of the disease. It was accepted practice for physicians to advise hypertensive patients to adopt a calm, unhurried way of life and to see problems in their proper perspective. The fact that hypertensive patients had problems in special spheres of activity about which they had little insight was often unappreciated. The physician might be of considerable help in recognizing and dealing with these conflicts. The concluding statement credited to Dr. Sokolow is one which is true in regard to almost any disease. He stated that a strong, confident, optimistic physician who used explanation and reassurance about daily variations in pressure and upon whom the patient could lean might, by his very presence, relieve symptoms, decrease invalidism, and permit the patient to live a happier life.

[1] *Am. J. Med.*, November, 1954.
[2] *Am. J. Med.*, November, 1954.

MYASTHENIA GRAVIS IN THE NEWBORN.

ALTHOUGH *myasthenia gravis* is usually a disease of adult life rather than of childhood or of old age, it does occur in children. Of the cases occurring in children a small and important group are in the newborn. These in turn are of two types—a transitory form in the children of myasthenic mothers and a true congenital form. According to S. Kibrick,[1] infants with the transitory form show signs of severe disease at birth and require treatment with neostigmine. Otherwise they are likely to die. If, however, they survive for two to four weeks (with or without treatment) they become normal and treatment is no longer required. Common signs of this condition are generalized atonia, inability to suck and difficulty in swallowing, a weak cry, regurgitation of feeding, a mask-like facies and inability to keep the eyes open. In some cases these signs are not noted for two or three days after birth; it is suggested that this delay may be due to the persistence of the effects of maternal medication. Infants with the second type of condition, which seems to warrant the term congenital myasthenia, are usually born of non-myasthenic mothers. The signs of disease are less severe than in the transitory type. Kibrick states that bilateral ptosis has been noted in all cases, and other frequent signs are a weak cry and difficulty in feeding. As the children grow older, various signs of muscular weakness such as indistinct speech, easy fatigue and frequent falling are noted. Kibrick suggests that any infant with unexplained limpness, inability to suck, a feeble cry and, particularly, evidence of cranial nerve palsy should be given a test dose of neostigmine, which, as in the adult and sometimes supplemented with ephedrine, is used both in diagnosis and in treatment. The test dose recommended for the newborn is 0·125 milligramme of neostigmine methylsulphate given subcutaneously. Oral dosage of the drug ranges from 1·0 to 3·75 milligrammes of neostigmine bromide. For transitory myasthenia of the newborn Kibrick suggests that replacement transfusion may cure the infant by removing the circulating substance which is postulated to inhibit the release of acetylcholine at the myo-neural junction.

[1] *Pediatrics*, October, 1954.

EDWARD MELLANBY.

SIR EDWARD MELLANBY, known the world over as a medical investigator of the highest rank, died on January 30, 1955, at the age of seventy years. The honours that were showered upon him by the Sovereign, by universities in many countries and by learned societies were attempts to show appreciation of his research and of the benefit which through it he conferred on mankind. He had one of those rare first class minds of clear and incisive quality that are of immense significance in science. Wherever in the whole world further work is done on vitamins, there shall the name of Edward Mellanby be recalled and had in honour.

Abstracts from Medical Literature.

RADIOLOGY.

X-Ray Demonstration of Cervical Nerve Root Avulsion.

J. E. WHITELEATHER (*Am. J. Roentgenol.*, December, 1954) states that cervical nerve roots may be avulsed from the cord or torn apart in the intervertebral foramina or immediately outside by injuries that impose severe traction upon the brachial plexus. Such injuries may occur when strong downward pressure is applied to the shoulder with the head fixed or pushed in the opposite direction. The degree and extent of the motor and sensory deficit, of course, depend upon the number of roots involved and on whether all fibres of a root are avulsed or separated. If roots are avulsed, or torn within the foraminal canal, the injury is irreparable. However, if it should be located just outside the foramen, there is some hope that neurorrhaphy may be successfully carried out. Myelography offers a quick, relatively easy and accurate method of demonstrating avulsion or root tear within the foramen. By exclusion, the remaining lesions could be considered extraspinal. Standard cervical myelographic technique is employed with six millilitres of warmed "Pantopaque". The spinal needle is left in place to facilitate removal of the oil. Recently the author has used nine millilitres on some occasions, finding that this is easier to manipulate, fills the theca better and is no more difficult to remove. The abnormalities are of two types and both are usually seen in the same patient. The first and most arresting is the presence of one or more extradural collections of oil in pockets or pseudodiverticula. These lie alongside or project into the intervertebral foramina and represent a tear or rent in the dura left by the avulsed root. A few fibres may still remain in these pockets. The second is less conspicuous but indicates a complete avulsion. The dura has healed, sealing the rent and leaving a smooth straight contour or only a tiny notch where the normal root shadow should be.

X-Ray Studies of the Small Intestine in Sprue.

R. H. MARSHAK, B. S. WOLF AND D. ADLERSBERG (*Am. J. Roentgenol.*, September, 1954) made a clinical and radiological study of 40 patients suffering from sprue over a period of one to seventeen years—three patients over more than fifteen years, eight over more than five years, twenty over more than three years, and ten over one year or less. The chief symptoms and signs included steatorrhœa, weight loss, anæmia, oral lesions, hypocalcæmia, hypoproteinæmia, flat vitamin *A* and glucose tolerance curves, and normal findings from pancreatic enzyme studies. The patients were observed radiologically before and after treatment in the hospital, though the vast majority had received some form of antianæmic therapy before coming under control. Examination of three patients revealed a normal small intestinal pattern. The remainder exhibited two distinctive patterns. The first and most characteristic was observed in 70% of the patients in this series and consisted of dilatation most prominent in the mid and distal parts of the jejunum, segmentation, large (four to ten centimetres long) barium-filled loops of ileum best visualized during the evacuation of the barium meal, thickening of the mucosal folds and the presence of hypersecretion of an altered quality. The authors state that while each of these features may occur in a wide variety of disorders, they are more frequent in and more pronounced in sprue. In the second pattern, segmentation is marked, early and persistent and is present throughout the small intestine; secretions are pronounced, and dilatation is slight to moderate. This latter pattern occurred in only 10% of the patients in this series and is also observed on occasion in those conditions and diseases simulating sprue — namely, nephrosis, hyperthyroidism, cirrhosis, pancreatic steatorrhœa, pellagra and other deficiency states. Radiological evidence of improvement in the sprue pattern after steroid therapy was significant in three patients to the present time and in four patients who have been treated for many years with anti-anæmic therapy.

Bleeding Lesions of Gastro-Intestinal Tract in Infants and Children.

JOHN R. HODGSON AND ROGER KENNEDY (*Am. J. Radiol.*, October, 1954) state that the most common causes of bleeding from the gastro-intestinal tract in 246 infants and children seen at the Mayo Clinic were chronic ulcerative colitis, polypi of the colon, Meckel's diverticulum and intussusception. The source of the bleeding was undetermined in 19 cases. In this series the most common cause of gastro-intestinal bleeding from birth through one year of age was intussusception; from two to six years of age, polypi of the colon; from seven to fifteen years, chronic ulcerative colitis. Some of the problems of radiological diagnosis of these and other causes of gastro-intestinal bleeding in infants and children are discussed.

PHYSICAL THERAPY.

Radioactive Iodine in Malignant Melanoma.

R. C. KORY, R. G. TUCKER AND G. R. MENEELY (*Am. J. Roentgenol.*, July, 1954) state that tyrosine is metabolized by epidermal melanoblasts to form melanin. If diiodotyrosine labelled with radioactive iodine could react as a substrate for the melanoblastic enzymes it might be possible to incorporate radioactive iodine in the melanomatous tissue thus delivering a large dose of β radiation to the malignant cells. The present study was undertaken to explore the possible usefulness of I^{131} as a therapeutic agent in this disease. Nine male patients were studied, of whom one died before a therapeutic dose could be given. The remaining eight received oral doses of 50 to 65 millicuries of radioiodine as sodium iodide. Seven of these eight patients also were given daily doses of thyroid extract in order to block the uptake of radioiodine by the thyroid. The results were disappointing. In no case was there any evidence of selective uptake of radioiodine in the melanomatous tissue. None of the patients exhibited either subjective or objective evidence of benefit from the administration of radioiodine, and the average survival time after treatment was 4·4 months. All patients had either regional lymph node or distant metastases, and it was concluded that radioiodine in the treatment of malignant melanoma was completely ineffective.

Radiotherapy for Intrinsic Tumours of the Spinal Cord.

E. H. WOOD, A. S. BERNE AND J. M. TAVERAS (*Radiology*, July, 1954) present a study of 62 cases of intrinsic tumours of the spinal cord, of which only 39 were pathologically classified. Of these 39 tumours, 23 were ependymomata and 12 astrocytomata. Radiation therapy was administered to each of the 62 patients in the immediate post-operative period. The total dosage varied considerably. Often more than one course of treatment was given. In assessing results, the authors have taken as a guide the figures from the Mayo Clinic series of 34 patients with spinal tumours in which the average survival was six years with surgery alone. In this present series of 39 proven cases treated by surgery and radiotherapy, 25 patients survived beyond the six-year limit. In addition, radiation therapy was found to increase recovery of function following surgical treatment of spinal glioma and usually inhibited advancement of disability if recurrence had taken place. Pain was almost invariably relieved. The authors state that experience has produced a trend towards conservative surgery, which usually consists of decompression by laminectomy, evacuation of areas of cystic degeneration in the case of intramedullary tumours and removal of easily accessible portions of tumours in the case of lesions of the lumbo-sacral area. There is also a trend towards more intensive radiation therapy as the tolerance of the normal tissue has become known. It is concluded that post-operative irradiation of all patients with primary intrinsic spinal cord tumours is justified.

Radiation Treatment of Carcinoma of the Corpus Uteri.

W. E. COSTOLOW, J. F. NOLAN, G. C. BUDENZ AND L. DU SAULT (*Am. J. Roentgenol.*, April, 1954) state that a study was undertaken to ascertain the relationship between the clinical results of radiation therapy and the dose delivered in the treatment of a series of 248 patients suffering from carcinoma of the body of the uterus. These patients were treated between 1933 and 1951 by intrauterine and intravaginal administration of radium, followed by external irradiation unless a hysterectomy was to be performed. In the early years the intrauterine radium consisted of two tubes in tandem, but in

recent years the multiple capsule packing technique has been used. The overall five-year recurrence-free rate was 36%, which was not regarded as satisfactory. A point "C" was chosen for estimation of dosage. This point lay 5·0 centimetres above and 2·5 centimetres lateral to the external os, and dosage was calculated from the known distribution of the radium. A figure was fixed for a minimal desirable tumour dose, which varied of course with the overall time of treatment, and a graph was constructed with the dosage given to each patient in relation to this minimal dose. It was found that there was a definite relationship between clinical success and the dose delivered. Above the line 72% of successes were found, and below it were 61% of the failures. This relationship was found also to hold when the patients were divided into three groups according to clinical stages of the disease.

Radiotherapy for Cushing's Syndrome.

J. F. P. SKRIMSHIRE (*Lancet*, February 5, 1955) reports the treatment of six patients suffering from Cushing's syndrome. Deep X-ray therapy was given to the pituitary gland with a high-voltage unit. A maximum dose of 4000*r* to 5000*r* was given in twenty-eight to thirty days, the resulting skin dose being about 3000*r*. One patient died of cerebral hæmorrhage five months after completion of treatment, but had shown improvement in relation to the Cushing's syndrome. Of the five surviving patients four improved considerably; one improved temporarily, the condition subsequently becoming non-progressive. The authors consider that controlled Cushing's syndrome is preferable to controlled adrenal insufficiency, and that radiotherapy should be used before surgery is contemplated; surgery can be used later if necessary.

ORTHOPÆDIC SURGERY.

Parosteal (Juxtacortical) Osteogenic Sarcoma.

LEONARD A. DWINNELL, DAVID C. DAHLIN AND RALPH K. GHORMLEY (*J. Bone & Joint Surg.*, July, 1954) have reviewed 15 cases of parosteal osteogenic sarcoma in the files of the Mayo Clinic. The ages of patients varied from twelve to forty-six years, the largest group being in the fourth decade. In the cases included in this study the tumour did not seem to arise in a pre-existing benign lesion. The authors believe that this lesion is malignant from its inception. The tumour has a marked predilection for the lower end of the femur, is attached to the cortex by a broad base, is ordinarily believed to be an atypical osteochondroma by the radiologist, and frequently confuses the pathologist by its subtle evidences of malignancy. The presenting complaint in each instance was the presence of a mass. On X-ray examination the typical tumour appeared as a dense lobulated mass attached to the metaphyseal region and sometimes also to the epiphysis of a long bone. All the tumours were attached by a broad base, and all but one were described as being sclerotic. In most cases, there were also areas of lesser density scattered throughout the tumour. Its borders were ordinarily sharply defined, but in some cases the border was indistinct in those areas where it blended with the surrounding tissue shadows. As the tumour grew, it sometimes extended along the shaft, but it almost always tended to surround the bone. In some instances the latter process was complete, the shaft of the bone then running through the tumour. Reactive periosteal new-bone formation and medullary involvement were conspicuous by their absence in the primary lesions. In differential diagnosis it is necessary to distinguish the tumour from ordinary osteogenic sarcoma, which is of a high degree of malignancy, grows rapidly and almost always involves medulla. It may be confused with osteochondromata and exostoses, which have a broad base in some instances, but the cortex of the bone is continuous with and forms part of the tumour. Their periosteal covering is intact, and muscle and fat are not trapped within their substance. The core of these tumours is continuous with and identical with the underlying medullary bone. The cartilage present is in the form of a cap rather than scattered throughout the tumour. Malignant change is rare in these lesions and usually occurs in the form of pure chondrosarcoma. Chondrosarcoma, predominantly on the surface of bone, or periosteal chondroma, must be differentiated from parosteal sarcoma. Such cartilaginous lesions lack the prominent bony and fibrous components of juxtacortical osteogenic sarcoma.

The Muscle-Pedicle Bone Graft in Hip Fusion.

JOE B. DAVIS (*J. Bone & Joint Surg.*, July, 1954) has operated on nine patients whose ages ranged from twenty-three to seventy-two years and attempted to fuse a hip joint which was the site of degenerative arthritis. In one case fusion did not occur—it is believed because of technical error. In all other cases fusion occurred; the average time of bony ankylosis was thirteen weeks. The author found that the hip joint, with the adjacent ilium, lends itself well to transplantation of bone with an attached muscle pedicle through which the periosteal blood supply can be maintained. An anterior Smith-Petersen incision is started 7·5 centimetres behind the anterior superior iliac spine; this is extended along the anterior crest of the ilium to the anterior superior iliac spine and is curved downward to end near the base of the greater trochanter. The incision is then carried to the iliac crest, and the space between the sartorius and the *tensor fasciæ latæ* muscles is identified. The sartorius muscle is detached from its origin, secured by suture and retracted out of the wound. The superior rim of the anterior crest of the ilium is then approached. By subperiosteal dissection, the aponeurosis of the abdominal muscles and the *iliacus minor* are separated from the superior and medial surface of the ilium. This preparation of the field permits removal of a bone graft and leaves the external muscles intact. By means of a vertical osteotomy at the posterior limb, the anterior 6·4 centimetres of the crest of the ilium are removed to a depth of 2·5 to 4·0 centimetres, depending on the need in the particular case. During this part of the procedure the osteotome is, of course, directed from inward to outward. After the bone has been freed, it has a muscle pedicle consisting of the *tensor fasciæ latæ* and the anterior fibres of the *gluteus medius* and *gluteus minimus*. By sharp dissection in the course of the fibres of the *gluteus medius* a muscle pedicle is developed. When hæmostasis has been assured, the direct head of the *rectus femoris* muscle is detached and the capsule is opened. The hip joint is then dislocated, and the cartilage is removed from the acetabulum and the femoral head. The femoral head is then reduced into the acetabulum, and the direct head of the *rectus femoris* muscle is reattached to its place of origin. In preparation of the site for placement of the graft, a slot is made in the ilium superior to the acetabulum, and a groove is made across the neck of the femur. The exact location of these incisions can be ascertained by placing the graft across the area it is to cover. An intertrochanteric osteotomy is then performed through the lower limb of the incision. The purpose of this procedure is to obtain optimum immobilization of the area of fusion. Adjustment of the position of the extremity as desired is facilitated through the osteotomy site. Care should be taken not to place the osteotomy site lower than the lesser trochanter because of the danger of non-union. Mechanical fixation at the osteotomy site should not be carried out. The osteotomy is performed before the graft is secured in order to avoid disturbance of the graft. After this procedure has been accomplished, the graft is secured by vitallium screws, one being placed through the graft into the ilium and another across the graft and the femoral neck. The screws will thus be at about a 90° angle to each other and fixation will be firm. Immediately after the operation the patient is placed in a standard hospital bed, and eight pounds of skin traction are applied to the affected extremity. This traction is continued for seven to ten days. At the end of this time the patient is placed in a double spica cast, long on the side operated upon and short on the well side. There are a number of advantages in waiting several days before applying the spica cast. One is that the patient can be cared for more easily after operation. Another is that the cast can be applied so that it is worn with more comfort. Still another advantage is the possibility of adjusting the position of the extremity more accurately through the osteotomy site when the paraphernalia of the operating room are not present. The original spica cast is maintained for six weeks. It is then changed to a double spica cast which extends only to the knee on each extremity. The patient is made ambulatory with walkers, parallel bars or crutches, depending on his physical prowess. Knee motion is maintained by physical therapy.

Congresses.

PAN-PACIFIC SURGICAL ASSOCIATION: SIXTH CONGRESS.

THE sixth congress of the Pan-Pacific Surgical Association was held in Honolulu from October 7 to 18, 1954.

The Pan-Pacific Surgical Association has a permanent office and officers in Honolulu, and every three years conducts a congress to which all surgeons of the Pacific nations are invited. The attractions of a week in beautiful Hawaii, as well as the benefits of a first-class scientific programme, combine to assemble surgeons from medical centres of the whole Pacific area and from many other schools in Canada and North America.

The first congress was held in 1929 and comprised about 14 registrants. That of 1954 brought together over 150 participants *plus* 700 registrants from 18 countries, who enjoyed a week of perfect weather, stimulating discussions and warm hospitality. The section of the programme allotted to general surgery was subdivided into subsections dealing with cardio-vascular, biliary, gastric, large bowel, thyroid, thoracic and breast surgery. Other sections concerned themselves with obstetrics and gynæcology, urology, oto-rhino-laryngology, orthopædics, ophthalmology and neurosurgery.

The congress began on October 8, with formal ceremonies, in which traditional Hawaiian welcomes were combined with official speeches and remarks by Governor Samuel W. King (Governor of the Territory), Dr. I. S. Ravdin (President of the Association), Dr. F. J. Pinkerton (Director-General) and official representatives of the United States Navy and Army, the American Medical Association and the American College of Surgeons. Next day a panel discussion dealt with "Problems of Intestinal Obstruction" and "Plasma Expanders". In the latter it was revealed that dextran derived from beet sugar, and already in fairly common use in Australia, caused a 100% blood volume increase and was fully metabolized and excreted. The amount to be administered was stated to be not more than 1000 millilitres in twenty-four hours, as otherwise it might prolong bleeding time as the result of a fall in blood fibrinogen content. If further infusions were required, blood itself should be employed. Allergic reactions could be anticipated from its use in 4% of cases. Small subendothelial and subendocardial hæmorrhages had been observed to occur in dogs, with which polygelatin had been the most innocuous of the plasma expanders under study. Blood plasma could not yet be rendered virus-free, and use of this expander had resulted in virus hepatitis on 25% of occasions in the recent Korean campaign.

The remainder of the scientific programme occupied a week's mornings, commencing with breakfast clinics at 7.30 a.m. and continuing from 9 a.m. to noon. Papers totalling 150 were presented.

Among the outstanding presentations were those concerned with strictures of the common bile duct; it was pointed out that while three-fourths of these were secondary to trauma incident to cholecystectomy, a few were due to damage to a single small artery arising from the supraduodenal artery, which might be the only blood supply to the duct. For melanomata, radical excision of the regional lymph nodes was advocated, whether they were palpable or not. Retrograde aortography was said to be an important diagnostic technique in cardiac failure of infants suffering from coarctation of the aorta or patent *ductus arteriosus*. It was reported that the immediate treatment of arterial injuries by ligation had been abandoned in the Korean War and replaced by prompt repair and reconstruction of the artery, with striking reduction in the amputation rate. Over 70 cases of aneurysm of the aorta treated by extirpation were described from one centre. Feeding by a plastic duodenal tube in which the feeding tube was inside the suction tube was advocated as a sequel to gastric surgery. Experience with pliable tubes of thinly woven "Nylon" fused with a thin sheet of non-reactive polythene for bridging aorta defects after excision of the aneurysm had revealed no difficulties in eight of ten cases. A new concept of the surgical attack on chronic ulcerative colitis, a greater knowledge of the ætiology of ileostomy dysfunction and the use of modern ileostomy appliances appeared to have done much more for the patients concerned than more conservative measures. It was stated that under "ideal" conditions ileostomy, colectomy and resection of the rectum could be carried out at one sitting. It was computed by another speaker that at least 12,000 thyrotoxic patients have been treated with radioactive iodine during the past fourteen years, with successful results in 86% of cases. A series of 23 patients with hyperparathyroidism was analysed; renal calculi or nephrocalcinosis overshadowed the clinical bone changes in frequency; advice was given to have blood calcium estimations performed at more than one laboratory if clinical and urinary evidence suggested the diagnosis. Other topics considered were the diagnosis of tumours of the neck by the use of radioactive iodine, the management of parotid gland tumours, the production of pericardial adhesions for the relief of coronary artery disease, the inclusion of block dissection of the internal mammary lymph node chain as part of the operation of radical mastectomy, and the management of pancreatic tumours and of acute pancreatitis. In a discussion concerned with the surgery of pulmonary tuberculosis, it was claimed that lobectomy for localized disease had resulted in a return of 90% of patients to normal activities. Subtotal œsophagectomy with anastomosis to the stomach brought up almost to the neck by a combined right thoracic cervical and abdominal approach was reported to be the only successful procedure for the cure of stenosing œsophagitis.

Space does not permit a synopsis of papers presented under the special subsections, but the same variety and quality of presentation were maintained.

The Australian medical profession was represented by about ten members from New South Wales, Victoria and South Australia, who contributed to the programme as follows: "Research Observations in the Course of Biliary Surgery", Dr. F. Rundle (Sydney); "Some Thoughts on Gastrectomy for Peptic Ulcer", Dr. I. Hamilton (Adelaide); "Acquired Tricuspid Stenosis: Medical Aspects", Dr. K. Maddox (Sydney); "Acquired Tricuspid Stenosis: Surgical Aspects", Dr. F. Mills (Sydney); "Anterior Resection of the Rectum", Dr. T. E. Wilson (Sydney); "The Swollen Leg and Stasis Ulceration", Dr. A. Sharp (Sydney); "The Modern Approach to Pancreatitis", Dr. K. W. Starr (Sydney); "Corneal Grafts in Australia" and "Confusion, a Disadvantage of Binocular Vision", Dr. T. A'B. Travers (Melbourne); "Angiography in the Management of Intra- and Supra-Sellar Tumours", Dr. R. A. Money (Sydney).

Further sessions were concerned with hospital management, staff relationships in hospitals and hospital accreditation, under the chairmanship of Dr. E. L. Crosby, Director of the American Hospitals Association, and one morning was given over to the showing of surgical films.

Dr. Arthur Allen, of Boston, was elected President of the Association, and Dr. I. Hamilton, of Adelaide, a Vice-President for the forthcoming three years, and the seventh session was scheduled for October, 1957.

British Medical Association News.

NEW SOUTH WALES BRANCH NEWS.

FOCLA Annual Post-Graduate Course.

THE annual post-graduate course arranged by FOCLA (Federation of Country Local Associations of the New South Wales Branch of the British Medical Association) will be held at Wollongong from March 14 to 18, 1955. All lectures will be held at Phillip House, Crown Street West, Wollongong. The scientific programme is as follows:

Monday, March 14: 9.30 a.m., inauguration; 10 a.m., official opening; 11.15 a.m., Dr. H. H. Lee, "Medical History of the Wollongong District"; 12.15 p.m., Dr. K. C. T. Rawle, "The Dr. R. D. Mulvey Memorial Oration"; 2.15 p.m., Dr. C. G. McDonald, clinical address; 3.30 p.m., Dr. A. J. M. Sinclair, "Psychotherapy in General Practice".

Tuesday, March 15: 9.15 a.m., Dr. H. K. Porter, "Don'ts of Gynæcology"; 10.45 a.m., Professor B. T. Mayes, "Could the Chapter on Eclampsia Disappear?"; 11.45 a.m., Professor H. Messel, "Atomic Medicine (1)"; 2.15 p.m., Dr. Leslie Parr, "Advances in Treatment of Rheumatic Diseases"; 3.30 p.m., Professor H. Messel, "Atomic Medicine (2)"; 8 p.m., visit to the steelworks.

Wednesday, March 16: 9.15 a.m., Professor L. Dods, "Pædiatric Problems"; 10.45 a.m., orthopædic panel; 11.45 a.m., Professor L. Dods, "Therapeutic Problems in Pædiatric Practice".

Thursday, March 17: 9.15 a.m., pre-operative and post-operative care panel (chairman, Dr. V. M. Coppleson); 10.45 a.m., Dr. R. A. Kern, "Practical Considerations in the

Management of Allergic Patients"; 11.45 a.m., Dr. V. M. Coppleson, "Surgery of the Rectum and Anus"; 2.15 p.m., Dr. R. A. Kern, "The Forties and the General Practitioner".

Friday, March 18: 9.15 a.m., Sir Hugh Poate, "The Thyroid"; 10.45 a.m., Dr. T. M. Greenaway, "Neurological Diagnosis in General Practice"; 11.45 a.m., Dr. I. D. Miller, "Head Injuries"; 2.15 p.m., chest clinic (Dr. K. G. Outhred, Dr. R. G. Epps, Dr. M. J. Flynn), "Chronic Bronchitis and Emphysema", lecture and demonstration.

A programme of social and recreational events has also been arranged. A golf cup which has been donated anonymously to perpetuate the name of the late Dr. Roy Mulvey will be played for on Wednesday, March 16. A dinner will be held at 6.30 p.m. on Thursday, March 17, at the Wollongong Leagues' Club (dress: dinner jacket).

The course secretary is Dr. J. D. Maude, Church Street, Wollongong. Telephone: B 1905.

Public Health.

MEASURES FOR THE PARTIAL PREVENTION OF DENTAL CARIES.

THE following report is published at the request of the Director-General of Health, Canberra.

At the thirty-seventh session of the National Health and Medical Research Council, held in Canberra in November, 1954, the Council approved the report of an advisory panel appointed to study and report on the influence of climate on fluid consumption and the relationship of climate to total fluoride ingestion. The advisory panel consisted of Professor W. V. Macfarlane, Dr. N. D. Crosby, Dr. N. E. Goldsworthy and Dr. E. H. Hipsley (secretary). The Council considered that some public health authorities would probably require information concerning safe levels of fluoridation of water supplies under varying Australian climatic conditions, and therefore it recommended that part of the report of the advisory panel dealing with these safe levels of fluoridation should be given publicity in THE MEDICAL JOURNAL OF AUSTRALIA. The full text of the report of the advisory panel on water consumption will be found in the report of the thirty-seventh session of the National Health and Medical Research Council.

Recommended (Provisional) Levels for Fluoridation of Water Supplies Related to Climate.

Climatic factors that could be presumed to have an effect on water consumption are air temperature, radiant heat gain, relative humidity and wind movement. Variations in relative humidity and wind movement between the different parts of practically all of the inhabited areas of Australia would in practice have no measurable influence on water consumption. Differences in radiant heat gain markedly affect water consumption; but as radiant heat gain depends on outdoor exposure to solar radiation, and as it is closely related to air temperature, no allowance need be made for it as a separate factor in estimating the water consumption for various climates. Therefore, the panel considered that in order to calculate the effect of climate on water consumption for the purpose of recommending levels of fluoridation, the only factor to be considered is temperature.

The panel considered that the main variation in fluid consumption would arise from environmental conditions experienced during the daylight hours. When a man is asleep in bed, not only do environmental differences tend to be minimized by the bedding used, but also owing to the absence of work and exposure to radiation and other factors, no great demands are made on the cooling mechanisms of the body. Moreover, the point at which the body in the resting condition markedly steps up the evaporative cooling process lies between 90° F. and 95° F. In consequence, it is believed that under normal social conditions involving daylight work, fluid consumption would be more closely related to daylight temperatures.

There are not in existence any convenient data showing mean daylight temperatures for Australia, but it is probable that the normal maximum temperature (figures for which are available) will vary in the same ratios as mean daylight temperatures.

The normal maximum temperatures for January and July, representing midsummer and midwinter respectively, are shown in Maps 1 and 2.

There is very little direct information regarding the fluid consumption of children in relation to climatic temperatures.

Other factors, such as climate and degree of activity, being equal, fluid requirements and calorie requirements are each directly related to age and body size. Calorie requirements for different ages and body sizes, and fluid requirements for one age and body size, have already been determined. A reasonable estimate of the fluid requirements for other age groups and body size can be made by varying them in the same ratios as the calorie requirements for these groups.

Adolph (1947) has estimated that resting fluid requirements of an adult living at the temperature of the hottest inhabited areas of Australia in January would be four quarts (4·8 litres). By calculating the fluid requirements in the same ratios as the calorie requirements it can be estimated that the fluid requirements of a child of eight years in these areas in January is approximately 3·2 litres.

In making estimates of the fluid consumption of children, it is important to take into account fluid consumed as milk. This is especially true in cold climates, where milk may constitute practically the whole of the visible fluid consumed. For the purposes of this calculation, the panel has assumed that the average child of eight years would consume 600 millilitres of milk per day. With these assumptions and using the scanty data from the literature, the panel has estimated the fluid intake (excluding milk) for a child of eight years to be approximately as shown in Table I.

TABLE I.

Approximate Estimates of Water Intake (Excluding Milk and Water in Food) for a Child of Eight Years.

Climate (Australia).[1]	Water Intake (Litres).	
	Maximum.	Minimum.
Hottest climate:		
Midsummer (over 90° F.) ..	2·5	1·0
Midwinter (over 60° F.)	1·5	0·75
Coldest climate:		
Midsummer (under 70° F.) ..	1·5	0·5
Midwinter (under 60° F.).. ..	0·75	0·5

[1] Temperatures are normal maximum.

From these estimates and on the assumption that the upper safe level of fluorine intake from all sources for children of eight years of age is 1·5 milligrammes of fluorine per day (McClure *et alii*, 1945), Table II has been prepared showing the recommended fluorine concentration

TABLE II.

Recommended Safe Concentrations of Fluorine in Water Supplies of Normal Maximum Temperature Zones in Australia.

Temperature Zone. (Degrees Fahrenheit.)	Fluorine. (Parts per Million of Water.)
95 to 100	0·4
90 to 95	0·5
85 to 90	0·6
80 to 85	0·7
75 to 80	0·8
70 to 75	0·9
65 to 70	1·0
60 to 65	1·1
55 to 60	1·2
50 to 55	1·3
45 to 50	1·4

related to the temperature zones (normal maximum temperature) shown in the maps (Figure I). Gaps in the data have been filled by interpolation.

Calculations from these data have shown that in any one locality the greatest seasonal climatic variation (between midsummer and midwinter) will not lead to a seasonal variation of the recommended fluorine level greater than 0·6 part per million of water. Experience in the United States of America, which, like Australia, has geographical areas with very variable midsummer and midwinter climates,

has shown that artificial fluoridation of water is satisfactory and safe if one value is adopted and maintained throughout the year. Therefore, the panel recommended that for each location the mean of the midsummer and midwinter temperatures shown in Figure I should be taken. The appropriate recommended fluorine level can then be determined by reference to Table II. This level should be maintained without variation throughout the year.

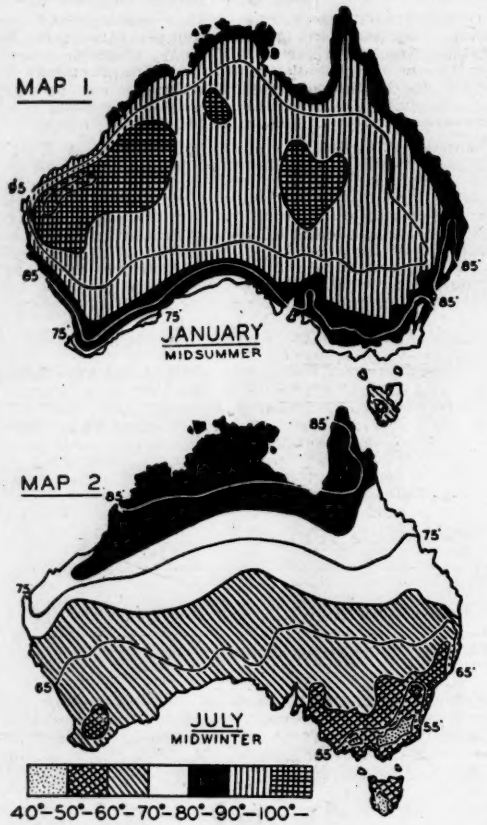

FIGURE I.

Ranges of normal temperatures. Adapted from "Atlas of Australian Resources", prepared and published by the Department of National Development, Commonwealth of Australia, 1953.

Thus for Sydney the midsummer normal maximum zone temperature is 80° F. and for midwinter it is 55° F., making the mean normal maximum 67·5° F. The recommended level of fluorine can be seen in Table II to be 1·0 part per million. Similarly other recommended levels (parts per million) can be calculated as follows: Hughenden and Darwin, 0·7; Alice Springs, 0·8; Hobart, 1·2.

The figures derived from these calculations agree closely with those recommended by Galagan (1953), who reviewed the effect of climate on controlled fluoridation levels in the United States of America. He concluded that 1·0 to 1·2 parts per million of fluorine may be considered optimal in zones comparable to the Great Lakes area (for example, Hobart), whereas 0·6 part per million may be considered to be equally effective in areas similar to Southern Arizona (for example, the hottest areas of Australia).

References.

ADOLPH, E. F. (1947), "Physiology of Man in the Desert", New York, Interscience Publishers, Inc.

GALAGAN, D. J. (1953), "Climate and Controlled Fluoridation", *J. Amer. Dental Assn.*, 47: 159.

MACFARLANE, W. V. (1953), unpublished data.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

PATHOLOGY.[1]

[From the *Sydney Morning Herald*, July 23, 1868.]

To the Editor of the Herald.

Sir: "Every living being" says a writer in "Household Words" "every man, woman and child endures a certain amount of sickness during life, for the alleviation of which medical knowledge and skill are required." But medical efficiency in the treatment of disease cannot be gained unless the young doctor bases all his subsequent studies on a thorough knowledge of the structure of the human body. This information can only be had by the use of the scalpel on the dead. The very notion is apt to send a thrill through every nerve of those unaccustomed to regard the subject in a philosophical light. But the terms are absolute—no dissection no knowledge. Hence the study of "Pathological Anatomy". But pathology cannot be learned in the dead-house. It can only be studied in the living subject. To associate the word pathos—sensibility, feeling—with the dissection of bodies that by no possibility can feel at all is a new edition of lucus a non lucendo. It is seeking the *morbid product* instead of the *morbid process*—it is as futile as cutting and carving and microscopically examining rotten potatoes in the hope of finding a cure and preventive of the potato rot. Instead of peering over the corruption *within*, attention might be better directed to the influence of agencies *without*, then *effects* could not be mistaken for *causes*, nor the *end* for the *beginning*. But

> By education most have been misled
> So they believe—because they were so bred.
>
> MEDICUS.

Hear this ye sophists who when life is gone
Peer into bodies motionless as stone
And there for *Sources of Disease* mistake
The *Final Flaws* your blunders help to make
Who yet in schools where no sharp critic scowls
Pass for "profound pathologists"—with owls
Why in the corpse fragmental ruin scan
Pathos exists but in the living man.

Correspondence.

MORTGAGING THE FUTURE.

SIR: An advertisement for the position of director of the department of anæsthetics at Saint Vincent's Hospital, Sydney, has been noted in the daily Press (*The Sydney Morning Herald*, December 1, 1954). The decision to make this appointment, the first of its kind in this city, and carrying the evident intention of improving the conditions of teaching and service in anæsthetics, is most warmly applauded.

It is noted with regret, however, that the salary offered, £2000 a year, is inadequate, even as a "commencing rate". The heavy responsibilities to be borne by the appointee demand at least the £3000 a year recently offered for a similar position at Saint Vincent's Hospital, Melbourne.

This letter is the outcome of a resolution by the New South Wales Section of the Australian Society of Anæsthetists and aims at giving this matter the publicity it deserves.

The society's objections to this appointment are broadly as follows: firstly, that the salary is inadequate, being in fact only slightly more than that paid at present to the anæsthetics registrar at Saint Vincent's Hospital, an appointment made largely for the purpose of preparing trainee anæsthetists for consultant status; secondly, that the device of enhancing such an inadequate salary with facilitated private practice may well prove to be a dangerous form of economy by a hospital administration.

[1] From the original in the Mitchell Library, Sydney.

A full-time appointee may well be encouraged in such further activity, preferably extramural, as may keep his outlook broad and his experience comprehensive, but he should not be placed virtually in economic dependence upon it. The society feels that such a position demands a first-class man, one fully trained, who, while he may be given the right of limited private practice, for such a reason as indicated, should be occupied with the organization and maintenance of a department of anæsthetics as an end in itself.

In the position under discussion, if the private practice is not made extensive, it will surely be a foregone conclusion that its incumbent will come to view the hospital appointment as a period of introduction for entry into private anæsthetic practice.

The pursuit of this short-sighted policy in fulfilment of a concept so enlightened is all the more regrettable since it must initiate a precedent for the underpayment of other directorships, in the same way as the "contemptuous pittances" accorded those working in the thoracic unit at the Royal North Shore Hospital have been the basis by which other similar payments in this city have been assessed. Such a policy lowers the standards of all the specialties and is indeed a direct reason why many graduates are discouraged—preferring to remain in general practice.

The society hopes that the authorities concerned with the inauguration of the appointment at Saint Vincent's Hospital will review their decision in this matter, keeping in mind the example of the Royal Alexandra Hospital for Children, whose advertisement for a director of anæsthesia at £2000 a year—with no private practice—has been unsuccessful in attracting any applicants either in Australia or abroad.

Yours, etc.,

ROBERT B. SPEIRS,
Honorary Secretary, New South Wales Section, Australian Society of Anæsthetists.

143 Macquarie Street,
Sydney,
January 24, 1955.

HOSPITAL SERVICE UNDER THE NEW SOUTH WALES MEDICAL PRACTITIONERS ACT.

SIR: An unfortunate situation has arisen in New South Wales out of the posting of recent graduates to country hospitals, in many cases against their wishes, while metropolitan hospitals which were anxious to employ them have been prevented from doing so.

A committee has been in existence for some years with the object of effecting liaison between the recent graduates and hospitals wishing to employ them. This committee consists of representatives of the Hospitals Commission, the Faculty of Medicine, the Post-Graduate Committee, and the New South Wales Branch of the British Medical Association. In the past it has functioned successfully and done a ticklish job very well. This year the same cannot be said. Without prior consultation the committee has reduced the allocation of recent graduates to metropolitan hospitals by about thirty and has drafted these to the country. The metropolitan hospitals concerned, during the post-war years when the number of doctors graduating was high, accepted more graduates than previously out of a sense of duty to these graduates; now, with additional commitments, they have come to require the additional numbers, and they have accepted the reduction with unconcealed embarrassment. Some of the doctors who have been railroaded are indignant, but they have been taken by surprise and caught on the hop and have found themselves scattered and without any leadership or spokesman.

Some examples will show how badly the machinery of hospital appointments has been creaking. Dr. A was a student at the Z hospital, and he applied for appointment to it. His position on the pass list was good enough for him to gain appointment to a teaching hospital, though only just, and the Z hospital applied to the committee for his services. When the appointments were made it was found that the Z hospital was allocated fewer graduates than it engaged last year and Dr. A was appointed to the Borrioboolagah Base Hospital.[1] Dr. A. objected that the Borrioboolagah Base Hospital is not recognized by the Royal Colleges as an institution affording the surgical, gynæcological and obstetrical training required of candidates for their diplomas and that the career he had hoped to embrace might be blighted; but no notice was taken of this objection, and he accepted the appointment lest by delay he should find himself even less favourably placed.

Another case shows how the committee's directions to graduates, uncoordinated as they were with the wishes of the parties concerned, were (not at all improperly) ignored by a hospital and graduates with ideas of their own, and how this produced injustice and hardship. Dr. B. and Dr. C were posted by the committee to the Y hospital, a large metropolitan hospital. The hospital immediately wrote to them and told them that their appointment had not been approved by its governing body. In fact the hospital had already engaged the services of Dr. D and Dr. E, who were favourably known to it, though they had been posted by the committee to the Currency Town Base Hospital[1] and the Bob's Flat Base Hospital[1] respectively. Dr. D and Dr. E took up their appointments at the Y hospital and Dr. B and Dr. C. found themselves unemployed. Dr. C by means of some good staff work was ultimately taken on at the Z hospital. I do not know what became of Dr. B, or who went to Currency Town and Bob's Flat. This was fortunate for Dr. C, but hard on Dr. A, whose position on the pass list was very much better than that of Dr. B, Dr. C or Dr. E.

Why should a committee formed with such a straightforward object have acted in the manner described? Can it be that the Hospitals Commission has determined to get the cheapest possible medical service for the country hospitals? Rather than create positions for fully registered graduates which would attract them to country hospitals needing their services, it has perpetrated through the committee a flagrant abuse of the power indirectly delivered to it by the *Medical Practitioners Act*, a section of which, making a year's service at a public hospital a prerequisite for full registration, has recently come into operation. I can only suggest that the other representatives on the committee, accepting membership of it out of a desire to help recent graduates, have been placed in a false position. The British Medical Association has always been opposed to the limitation of professional freedom. The Faculty of Medicine and the Post-Graduate Committee are concerned with the educational welfare of recent graduates, and their natural wish would be for as many as possible of them to serve in hospitals internationally recognized for the very high standard of their teaching and professional practice—indeed, the fact that the recent graduate in New South Wales receives only provisional registration must imply that the year of post-graduate hospital service is regarded by the legislature as an educational one and not as a form of civil conscription.

A similar position to that which has just arisen could arise again next year (or even this year after the deferred examinations) unless the profession exercises its ingenuity and goodwill on behalf of its most junior and least articulate colleagues. The teaching hospitals should be encouraged to assimilate as many recent graduates as they can.

Yours, etc.,

DOUGLAS ANDERSON.

185 Macquarie Street,
Sydney,
February 9, 1955.

[1] Pseudonymous designation.

THE TRAINING OF SPECIALISTS.

SIR: Attention is drawn to the announcement in the journal by the Post-Graduate Committee in Medicine in the University of Sydney of the second conference on graduate and post-graduate medical education which will be held in the Stawell Hall, 145 Macquarie Street, on the evening of Wednesday, March 2, 1955, at 8 p.m. The subject for discussion will be "The Training of Specialists".

This conference, as in the first conference, will take the form of a panel discussion on which the University of Sydney, the Post-Graduate Committee in Medicine, the Royal Australasian College of Surgeons, The Royal Australasian College of Physicians, the Royal College of Obstetricians and Gynæcologists and the College of Radiologists of Australasia will be represented.

In arranging for "The Training of Specialists" to be the subject of the second conference, the Committee hopes for a discussion on the trends of specialist education and our present methods of training in Australia generally and New South Wales in particular, especially in the light of recent changes and of newer methods in the United Kingdom and the United States.

The Committee has also completed a review of hospitals in New South Wales from the aspects of their potential in

[1] Pseudonymous designation.

the training of specialists and general practitioners. This has now been printed and copies are available on application to the Committee.

The Committee is of the opinion that this conference is of considerable importance in the development of graduate medical education in New South Wales and is anxious that it should be brought to the notice of as many of the bodies concerned and persons interested as possible. The Committee would also appreciate any comment or suggestions concerning these matters.

Yours, etc.,

V. M. COPPLESON,
Honorary Director, Post-Graduate Committee in Medicine in the University of Sydney.

131 Macquarie Street,
Sydney,
January 31, 1955.

DRIED PRESERVED GUINEA-PIG COMPLEMENT.

SIR: We have had the opportunity of testing 20 known Wassermann positive sera and 20 known negative sera using, as our complement, dried preserved guinea-pig serum prepared by the Research Laboratories at Beckenham, England, of Burroughs Wellcome and Company.

The method used was a half-volume standard Kolmer complement-fixation test ("Manual of Serologic Tests for Syphilis", 1949, Supplement No. 22, *The Journal of Venereal Disease Information*, United States of America). The 40 sera were first tested by our own routine method using azide-preserved complement prepared from our own guinea-pigs by the method of Richardson (*Lancet*, December 6, 1941). Two antigens were used—cardiolipin and cholesterolized alcoholic beef heart extract prepared in this laboratory. Commonwealth Serum Laboratories hæmolysin (glycerinated) was used, a 1:100 dilution of this being kept as the stock hæmolysin solution. The sheep cells were obtained fresh from the animal under non-sterile conditions, washed and used in a 2% suspension in normal saline. The 20 positive sera varied from weakly positive (titre 2·5) to strongly positive (titre 320).

The dried preserved guinea-pig complement was reconstituted following directions packed with it—namely, the serum was dissolved in two millilitres of distilled water, and one volume of this solution *plus* seven volumes of distilled water made the equivalent of a 1:10 dilution of fresh guinea-pig serum in saline. A 1:2 dilution of this complement was then prepared—making a final 1:30 dilution of complement—and preliminary hæmolysin and complement titrations were carried out—the end points being clear cut. Hæmolysin in the complement titrations and the routine tests was used two hæmolysin units in strength. These resulted in hæmolysin 1:1500 and complement 1:33 being used, comparable with our results using fresh guinea-pig complement.

The 40 known sera were then tested. The negatives were clear cut, the positives read easily, and there was no evidence of anticomplementary reactions.

Yours, etc.,

MARGARET GARNER.

Microbiological Laboratory,
New South Wales Department of Public Health,
Sydney.
February 1, 1955.

THE SURGICAL TREATMENT OF CARDIAC ISCHÆMIA.

SIR: The surgical treatment of cardiac ischæmia has lagged far behind that of other acquired diseases, such as mitral stenosis and constrictive pericarditis, and of congenital lesions, such as the patent *ductus arteriosus* and the tetralogy of Fallot. There are many reasons for this lag; most of the patients with cardiac ischæmia are at or past middle age, prognosis is difficult and no completely satisfactory operation has yet been devised. In England, just before the last war, the late Laurence O'Shaughnessy had carried out omento-cardiopexy with some promising but uncertain results. In America, Claude Beck has been working steadily at the problem for many years, but most surgeons and physicians have shown little interest in it. The whole subject was freely discussed by a special panel at the Congress of the American Association of Chest Physicians at Barcelona in October, 1954. There was general scepticism about all forms of treatment of cardiac ischæmia. Most of the panel voted that the anticoagulants and papaverine were of doubtful value, and there was general agreement that the best treatment at present consisted of modified bed rest and the administration of nitrites and analgesics. The panel's conclusion was that some form of surgery might eventually be the best treatment, and it urged thoracic surgeons to persevere with some of the operations that have already been tried and to work out new procedures.

In the final assessment of any operation it must be remembered that relief of anginal pain has followed simple thoracotomy, and it has also followed an operation, before any collateral circulation has had time to develop. These unexpected and unexplained results should encourage rather than deter both the patient and his doctor.

At this point it is worth while to review briefly the surgical procedures that have been tried.

1. One group of operations aims to improve the myocardial circulation by supplying fresh blood from a systemic artery: (*a*) the internal mammary artery is implanted into the myocardium, or (*b*) the artery is anastomosed to the coronary sinus.

2. A second group aims at improving the circulation by promoting collateral channels between the myocardium and some other organ or tissue, such as the omentum, the pectoral muscle, the lung, a pedicled skin flap or a jejunal loop.

3. The blood supply to the myocardium may be improved by inducing adhesive pericarditis by placing irritating substances in the pericardial sac—for example, asbestos and talc powder.

Another operation consists of ligating the great coronary sinus and cutting the nerves around the coronary arteries. A more exotic operation is the excision of an infarct.

It is clear that the whole surgical treatment of cardiac ischæmia is still experimental and of doubtful value, but cardiac ischæmia is so common and so devastating that we should constantly seek for some better treatment than is at present available.

Yours, etc.,

M. P. SUSMAN.

185 Macquarie Street,
Sydney,
January 4, 1955.

ADRENOGENITAL SYNDROME: REPORTS OF TWO ILLUSTRATIVE CASES.

SIR: I read Dr. Wait's recent article on the adrenogenital syndrome with interest. His Case I, a girl from whom an adrenal tumour was removed, is of particular importance, as she was said to have developed pubic hair in the first few months of life and her 17-ketosteroid excretion was not grossly elevated. This clinical picture is so much more likely to be due to adrenocortical hyperplasia that it serves as a salutary reminder of the suspicion with which all such cases must be viewed.

The recognition and successful removal of adrenal tumours from these two girls is indeed a matter for congratulation. However, Dr. Wait does not discuss pre-operative and post-operative care. Lest it should be felt that success comes easily in all such cases, I feel that their proneness to adrenocortical failure, in the form of sudden collapse or death, should receive emphasis. The necessity for adequate preparation with cortisone and for continuation of its use and readiness to treat signs of adrenocortical failure in the post-operative period are well documented in the literature and have been borne out by experience at the Royal Alexandra Hospital for Children.

Yours, etc.,

R. H. VINES.

135 Macquarie Street,
Sydney,
February 4, 1955.

MONGOLISM.

SIR: In your "Current Comment" in the journal of February 5 you refer to the diagnosis of "mongolism" in Mongolian races depending upon the "non-mongoloid" features. This is absurd. Actually the features of the mongoloid defective have little in common with those of normal children of the Mongolian race. The facial characteristics of the mongoloid defective have been described in detail, with drawings and photographs to illustrate the

differences between "mongoloids" and Mongolians, by Benda (1949) in his classical monograph.

Your statement that no alternative term has been put forward for "mongolism" is not quite accurate. Benda records a number of proposed alternatives and favours "congenital acromicria", a term which had been first proposed by Schuller in 1907 and again by Clift in 1922. He considers that the condition is due to a deficiency of that pituitary hormone, an excess of which in adult life produces acromegaly; and that the characteristic skeletal changes are those of underdevelopment of the small bones of the face, hands and feet.

Yours, etc.,

Hobart, Tasmania, February 11, 1955. J. R. V. FOXTON, M.B., B.S.

Reference.

BENDA, CLEMENS E. (1949), "Mongolism and Cretinism", Grune and Stratton, New York.

NOCTURNAL CRAMPS IN THE LEGS.

SIR: With reference to your article on nocturnal cramps in the legs in the issue of February 5, 1955, reporting relief from "Orthoxine", any quinine salt in a dose of five grains at bedtime gives an equally dramatic and reliable relief. Probably quinidine would have the same effect, in view of its soothing effect on the smooth muscle of the cardiac vessels or myocardium.

However, quinine is readily obtainable. It is very cheap, and there are no side-effects in this dosage. The only contraindication to its continuous use, which is seldom necessary, is pregnancy. In this age group the question of pregnancy does not arise.

Yours, etc.,

185 Macquarie Street, Sydney, February 7, 1955. BRIAN HAYNES.

Obituary.

ALLEN WILLIAM DAVID ROBERTSON.

WE have received from Dr. Colin Macdonald the following appreciation of the late Dr. Allen William David Robertson.

Few Victorian medical men can have had such varied interests over a long life as Allen Robertson, who died on November 9, 1954, in his eighty-eighth year. Press notices following his death recalled Dr. Robertson's effective chairmanship of the Australian Board of Cricket Control during the bitter bodyline controversy, with the hallowed Marylebone Cricket Club's tradition of sportsmanship thrown into critical question by the Australian Board. In other fields, his qualities of tenacity, patience and firmness of will stood him in equally good stead.

Allen William David Robertson had to work hard for everything he achieved. Born in the Riverina town of Deniliquin, where his father was manager of a local general store, he was early left an orphan. An aunt brought him to Melbourne, and until the age of thirteen years he attended King's College, a small private school in the suburb of Fitzroy, long since defunct. For the next seven years he worked in a Flinders Lane warehouse.

Young Robertson had by then determined to become a doctor, but, being without funds, decided that the best route to medicine was by way of pharmacy. To this end becoming apprenticed to H. C. Armstrong, a Melbourne chemist with branches in a number of large New South Wales country towns, Robertson served a term in each of those branches and thus acquired a wide knowledge of the countryside. This peripatetic existence did not affect his studies, for in 1895 he won the pharmacy gold medal, and matriculated in the University of Melbourne for entrance to medicine. He never told the full story of his pecuniary struggles to complete the course, but they must have been great, for towards the end of it finances became so straitened that he was forced to accept, for twelve months, a position as relieving pharmacist in a lonely outback town in Western Australia. Robertson had reached the age of thirty-six years on graduating M.B. in 1903, being placed ninth in a good year which included such well-known names as Douglas Stephens, B. T. Zwar and Harvey Sutton. In the latter part of the course he was resident in Queen's College, for whose distinguished master, Dr. E. H. Sugden, he always retained a high regard. Sugden of Queen's, MacFarland of Ormond, and Leeper of Trinity were the three great men who, in their several ways, brought to Melbourne the best traditions of British university life and laid the foundations of that residential collegiate system of which Melbourne is rightly proud. Sugden, a lovable Yorkshireman of wide humanity and culture, and world famous as a Shakespearian scholar, exercised a profound influence on resident undergraduates at Queen's during his long mastership.

Robertson served terms as resident medical officer at Saint Vincent's and the Women's Hospital, and then was for two years superintendent of the Austin Hospital at Heidelberg, in those years a semi-rural suburb of much beauty. It was here, on the banks of the Yarra River, that Tom Roberts, Arthur Streeton and Charles Conder had first painted the real Australian sunlight and shadow. Afterwards, Robertson bought the general practice of Dr. Walter McGibbon, in Brunswick Street, Fitzroy, remaining there for fifteen years, when he became a Collins Street consultant in diseases of women. During the Brunswick period he was a demonstrator in anatomy, and held for three years a research scholarship in anthropology, under Richard J. A. Berry, who had come from Edinburgh a few years before to the chair of anatomy. Berry had an especial interest in the skull and brain, and it was he who directed Robertson's attention to the craniometry of Australian, Tasmanian and New Guinea aborigines, for a thesis on which subject he was awarded the M.D. in 1910. He became a Fellow of the Royal Australasian College of Surgeons in 1928. Robertson wrote a paper on the anatomy of the sinu-ventricular system of the heart, and made occasional contributions on diseases of women, but claimed little distinction to spoken or written artistry, and developed no outstanding reputation as a clinical teacher. As a gynæcologist he was regarded as sound and sure, an innate caution forswearing risks of any kind. In 1910 Robertson was one of two honorary midwifery surgeons elected to the Women's Hospital; the other was the late J. H. Nattrass. They were the first appointees under the electoral college system generally adopted in Melbourne teaching hospitals today; previously, all appointments had been made on the votes of the hospital subscribers, a system

possessing obvious demerit, and the cause of much heart-burning in unsuccessful applicants. At that time the senior surgeons at the Women's Hospital included M. U. O'Sullivan, Rothwell Adam and Felix Meyer, all impressive personalities long to be remembered in Melbourne. Robertson advanced to the chairmanship of the staff in 1928, held until his retirement in 1931. But over the succeeding twenty-three years he remained sincerely attached to the Women's Hospital and was present at its annual meeting only a few weeks before his death, keenly attentive to the proceedings. Friends realized then that his life was drawing to a close, although few understood that he was approaching ninety years of age.

Convinced that a medical man's first loyalty should be to the British Medical Association, Robertson gave devoted service as a councillor of the Victorian Branch, which he had represented at the International Medical Congress in London in 1911. This was the great meeting presided over by Sir Thomas Barlow, and Robertson was profoundly impressed by that Nestor of British medicine who died ten years ago, just a few months before his one-hundredth birthday. As a senior vice-president, only ill health prevented Robertson from attaining the highest position in the Victorian Branch, his place as President in 1935 being taken by the late Major-General Rupert Downes. He was for nearly thirty years a representative of the graduates in medicine on the Standing Committee of Convocation of the University of Melbourne.

Robertson's interests ranged widely beyond the purely professional, not the least being cricket and its administration. An enthusiast for the game from childhood, he became president of the Melbourne University Cricket Club in 1914 and, also in that year, the club's delegate to the Victorian Cricket Association. Five years later he was appointed one of the three Victorian representatives on the Board of Control, taking the place of Dr. Ramsay Mailer; so greatly did he value this association with international cricket, that he remained thereon until three months before his end. In 1930 Robertson was elected Chairman of the Board, succeeding R. A. Oxlade, little suspecting then that two years later the combined efforts of a young Nottingham miner and an inflexible Wykhamist and Oxonian would light a fiery cross from Leeuwin to Cape York because of their grim determination to wrest the ashes from Australia. Robertson could not bring himself to accept any particular malignity in the fast bowler—of whose accuracy and beautiful rhythm he was an admirer—the dour captain or other members of the M.C.C. team. Many believe that it was in considerable part his wise and tempered counsel which resolved the unhappy conflict of ideas in a way considered the most satisfactory possible. Today's president of the Victorian Cricket Association expressed the opinion, on learning of Robertson's death, that his work for Australian cricket could never be repaid.

Possibly it was in the Wallaby Club that Allen Robertson obtained his greatest contentment. Here was a haven free from professional and business stresses, and he knew how very highly he was esteemed by all the members of this famous walking club, which for more than sixty years has included some of the leading doctors of Melbourne. It was, indeed, founded by a doctor, Louis Henry, who was also the founder of the British Medical Association in Victoria. Henry and Robertson shared a friendship broken only by the former's death in 1924. Robertson became secretary of the Wallaby Club in 1909, and his services were so highly regarded that he continued in the office for forty-five years, in that long time doing much to build the club's fine tradition of friendliness, and to maintain its happy and simple character. Its very existence is said to be a monument to Allen Robertson.

Allen's early training in warehouse and pharmacy made him a good man of business. He acquired pastoral interests, first in eastern Riverina, then nearer to Melbourne. He was one of the doctors' partnership which built Lister House in Collins Street, a handsome red-brick building, as attractive and functional today as when it was built forty years ago.

Allen Robertson is survived by his wife, whom, as Lydia Terry, he married in 1906. There were no children.

Throughout his long life, Allen Robertson remained a good figure of a man; with keen blue eyes, a fresh complexion and invariably a well-groomed appearance that until just before his death greatly belied his age. During a lifetime varied in experience and interests, he was a well-respected figure in Melbourne, and the Royal Women's Hospital is proud to add his name to its roll of merit.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE following notes relate to the Australasian Medical Congress (British Medical Association), Ninth Session, which is to be held at the University of Sydney from August 20 to 27, 1955.

Membership.

Membership is open to all members of the British Medical Association. Application forms for membership are available from the Honorary Local Secretary of Congress in each State. These are as follows: New South Wales: Dr. M. S. Alexander, 135 Macquarie Street, Sydney. Victoria: Dr. C. H. Dickson, Medical Society Hall, 426 Albert Street, East Melbourne, C.2. Queensland: Dr. D. A. Henderson, Ballow Chambers, Wickham Terrace, Brisbane, B.17. South Australia: Dr. L. Bonnin, 63 Palmer Place, North Adelaide. Western Australia: Dr. S. E. Craig, 7 Malcolm Street, Perth. Tasmania: Dr. K. S. Millingen, 178 Macquarie Street, Hobart.

Inaugural Meeting.

The inaugural meeting of Congress will be held in the Sydney Town Hall on the evening of August 22, 1955, when the President's address will be delivered by Sir Archibald Collins, Kt., D.S.O., M.C., M.B., Ch.M., F.R.A.C.P.

The Henry Simpson Newland Oration.

The Henry Simpson Newland Oration will be delivered in the Great Hall of the University of Sydney on the evening of Thursday, August 25, 1955, by Dr. Louis H. Bauer, Secretary-General of the World Medical Association, and a past president of the American Medical Association.

Vice-Presidents of Congress.

The following have been nominated as Vice-Presidents of Congress by the Branches of the British Medical Association throughout Australia and New Zealand: New South Wales, Dr. W. F. Simmons; Victoria, Dr. H. G. Furnell; Queensland, Dr. H. S. Patterson; South Australia, Dr. R. L. Thorold Grant; Western Australia, Dr. B. W. Buttsworth; Tasmania, Dr. T. C. Butler; New Zealand, Dr. J. C. Mercer.

Plenary Sessions.

Five plenary sessions will be held. The subjects will be "Cancer", "The Control of Infectious Diseases", "Rehabilitation", "The Use and Abuse of Hormones in Medical Practice" and "Industrial and Occupational Hazards to Health". The session on "Cancer" will be held from 9.30 a.m. to 12.30 p.m. on Tuesday, August 23, and the speakers will include Sir Stanford Cade, Professor E. S. J. King, Sir Peter MacCallum and Dr. C. E. Eddy. The other four sessions will be held on the afternoon of August 23, those on "The Control of Infectious Diseases" and "Rehabilitation" being held simultaneously from 2 to 3.30 p.m., and those on "The Use and Abuse of Hormones in Medical Practice" and on "Industrial and Occupational Hazards to Health" from 3.45 to 5.15 p.m.

Scientific Papers.

The following decisions of the Executive Committee are repeated for the benefit of members and intending members of Congress who desire to present papers to the meetings of sections. (i) Offers of papers will be received by the honorary secretaries of sections until March 31, 1955, and authors must indicate at least the title of the proposed paper with a precis of its subject if the completed paper is not submitted. (ii) Intending speakers will be notified not later than April 30, 1955, whether their offer of a paper has been accepted or rejected. (iii) Three copies in final form of papers accepted (in addition to the author's copy), typed in double spacing on one side of the paper only, complete with illustrations, together with a note of visual aid requirements, must be in the hands of the appropriate secretary of section by June 30, 1955. One copy will be for the chairman of the meeting at which the paper is to be presented, one for the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, and one for the lay Press liaison officer.

Under the rules of the Congress no paper can be taken as read, and unless it is read a paper does not form part of the proceedings of Congress.

All correspondence to honorary secretaries of sections should be addressed to the Congress office, 135 Macquarie Street, Sydney.

Social Functions.

The Congress dinner will be held on the evening of Wednesday, August 24, 1955. A ball, at which the hosts will be the members of the New South Wales Branch of the British Medical Association, will be held on the evening of Friday, August 26, 1955.

Exhibition of Hobbies.

An exhibition of hobbies will be an incidental but attractive feature of the Congress.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Lecture at Balmoral Naval Hospital.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that Dr. J. Witton Flynn will give a lecture on "The Principles in Diagnosis and Treatment of the Common Diseases of the Skin" on Tuesday, March 8, 1955, at 2 p.m., at the Balmoral Naval Hospital. Clinical cases will be shown after the lecture. Members of the medical profession are invited to be present.

Parts I and II Courses for Diploma in Dermatological Medicine.

Part I course of lectures for the diploma in dermatological medicine will begin in Sydney on May 16. Lectures will be held in the afternoons only and will extend over a period of twelve weeks, concluding on August 5. Part II course for this diploma will begin on August 15, consisting of afternoon lectures, and will continue until February, 1956. Fees for attendance are Part I course £31 10s., Part II course £63. Those interested in enrolling for these courses or obtaining further details concerning the diploma are requested to communicate with the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney, at an early date. Telephones: BU 4497-8. Telegraphic address: "Postgrad, Sydney."

Conference on Graduate and Post-Graduate Education.

The panel for the conference on "The Training of Specialists", to be held in the Stawell Hall, 145 Macquarie Street, Sydney, at 8 p.m. on Wednesday, March 2, 1955, is as follows: Dr. V. M. Coppleson, Director, Post-Graduate Committee in Medicine in the University of Sydney; Dr. Selwyn Nelson, Assistant Director, Post-Graduate Committee in Medicine in the University of Sydney; Dr. Julian Smith, Censor-in-Chief, Royal Australasian College of Surgeons; Dr. T. M. Greenaway, Censor-in-Chief, The Royal Australasian College of Physicians; Dr. S. Devenish Meares, Member of the Australian Regional Council, Royal College of Obstetricians and Gynæcologists; Dr. A. R. Colwell, Immediate Past President, The College of Radiologists of Australasia.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 4, of January 20, 1955.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Reserve.

Appointments. — Graeme Alvin Robson (Surgeon Lieutenant-Commander, Royal Australian Naval Volunteer Reserve) is appointed Surgeon Lieutenant-Commander, with

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED FEBRUARY 5, 1955.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Acute Rheumatism	1(1)	6(3)	1(1)	..	..	..	..	..	8
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	7(6)	7(1)	..	..	..	..	1	15
Diphtheria	3(1)	3(1)	4(4)	1(1)	11(10)	..	..	..	22
Dysentery (Bacillary)	..	..	3(3)	..	3(2)	1	..	..	7
Encephalitis	..	2	1	1(1)	..	..	..	..	4
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	33(18)	46(26)	..	6(1)	2	..	..	..	87
Lead Poisoning	..	..	1	..	..	..	..	..	1
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	1	1	1(1)	..	1	..	..	..	4
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	6(3)	15(6)	11(4)	2	2(1)	..	..	..	36
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	18(8)	..	1	4(2)	1	..	..	24
Salmonella Infection	..	..	..	..	4(1)	..	..	..	4
Scarlet Fever	13(3)	12(6)	4(2)	1(1)	2(1)	..	..	..	32
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	1	..	..	..	1
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	44(34)	26(22)	11(2)	5(5)	10(9)	5	..	1	102
Typhoid Fever	2(2)	1(1)	..	..	1	..	..	..	4
Typhus (Flea-, Mite- and Tick-borne)	1(1)	..	..	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from Northern Territory.

seniority in rank, of 12th August, 1947, dated 9th August, 1954. John Morton Copeland is appointed Surgeon Lieutenant, dated 14th October, 1954. Kenneth Frank Shepherd (Lieutenant) is appointed Surgeon Lieutenant, with seniority in rank, of 14th November, 1945, dated 19th January, 1954.

Ante-dating Seniority.—The seniority of Willoughby Sidney Sewell as Surgeon Lieutenant is ante-dated to 19th March, 1952.

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps.

The Short Service Commission granted to 3/40110 Captain J. M. McKenna is extended until 29th December, 1955.

1/8056 Captain G. L. Wadeson is retired, 22nd November, 1954.

Citizen Military Forces.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical).—2/130107 Captain C. B. Saunders is seconded whilst in the United Kingdom, 22nd October, 1954. To be Captains (provisionally), 18th November, 1954: F2/2804 Joan Hughes and 2/127892 Geoffrey Watkins Burgess.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—The provisional ranks of the following officers are confirmed: Captains 6/15245 B. A. Smithurst and 3/101828 H. A. Jenkins.

Central Command: Fourth Military District.

Royal Australian Army Medical Corps (Medical).—4/32052 Captain (provisionally) J. R. Richards relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (4th Military District) in the honorary rank of Captain, 12th November, 1954.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

1st Military District.—To be Honorary Captains, 18th November, 1954: Hugh James Falconer and Conrad James Primmer.

2nd Military District.—To be Honorary Captain, 22nd November, 1954: John Desmond Egan.—(Ex. Min. No. 1—Approved 12th January, 1955.

ROYAL AUSTRALIAN AIR FORCE.

Air Force Reserve: Medical Branch.

The appointment of the following officers is terminated: Flight Lieutenants (Temporary Squadron Leaders) J. C. A. Dent (261602), R. C. Gill (261482), R. W. Hazelton (261261), 29th September, 1954; Flight Lieutenants G. A. Levinson (297422), 6th March, 1954, L. A. Commins (264588), A. M. Hill (4366), A. W. McLaren (264591), 29th September, 1954.

Medical Appointments.

The following have been appointed members of the Council of the Queensland Institute of Medical Research for the ensuing triennial period: Dr. A. Fryberg; Dr. J. I. Tonge, nominated by the Minister to represent the Department of Health and Home Affairs; Dr. A. D. D. Pye, nominated by the Brisbane and South Coast Hospitals Board; Dr. G. C. Taylor, nominated by the controlling body of the Mater Misericordiæ Hospitals; Dr. W. H. Steel, nominated by the Queensland Branch of the British Medical Association.

Dr. T. W. Murray and Dr. H. P. Smith have been appointed medical officers in the Mental Hygiene Branch of the Department of Health, Victoria.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Jelihovsky, Tatiana, M.B., B.S., 1953 (Univ. Hong Kong), Royal Prince Alfred Hospital, Camperdown, New South Wales.

Merrifield, Alan John, M.B., B.S., 1954 (Univ. Sydney), Canberra Community Hospital, Canberra.

Taylor, Neville, M.B., B.S., 1953 (Univ. Sydney), 21 Liverpool Road, South Strathfield, New South Wales.

Deaths.

THE following death has been announced:

HOLLYWOOD.—James Joseph Hollywood, on February 14, 1955, at Sydney.

Diary for the Month.

MARCH 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MARCH 2.—Western Australian Branch, B.M.A.: Branch Council.
MARCH 4.—Queensland Branch, B.M.A.: General Meeting.
MARCH 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MARCH 11.—Tasmanian Branch, B.M.A.: Branch Council.
MARCH 11.—Queensland Branch, B.M.A.: Council Meeting.
MARCH 12.—Tasmanian Branch, B.M.A.: Annual Meeting.
MARCH 14-18.—New South Wales Branch, B.M.A.: FOCLA Post-Graduate Course at Wollongong.
MARCH 14.—Victorian Branch, B.M.A.: Finance Subcommittee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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